

























**DISEASES OF CHILDREN**





# DISEASES OF CHILDREN

EDITED BY

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AN AUTHORIZED TRANSLATION FROM "DIE DEUTSCHE KLINIK"

UNDER THE GENERAL EDITORIAL SUPERVISION OF

JULIUS L. SALINGER, M.D.

WITH THIRTY-FOUR ILLUSTRATIONS IN THE TEXT



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## INTRODUCTION

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HENOCH has thought that pediatrics should constitute the culmination of the student's work in medicine. Many diseases, although deviating in essential points, have great resemblance to the adult types. The student is therefore familiar with much, and only the special infantile diseases require particular investigation. This work consists of a series of monographs arranged so as to form a systematic treatise. The affections which do not differ from the adult type are here omitted; they will be found under their respective headings in the other volumes.

In the translation an attempt has been made to adhere as closely as possible to the style and phraseology of the German authors (a difficult task in many of these classic chapters). It is hoped that the book will fill a need and occupy the same place in English that it has acquired in the original.

JULIUS L. SALINGER.

1919 SPRUCE STREET, PHILADELPHIA.



## EDITOR'S PREFACE

AMERICAN pediatric literature has no lack of text-books. Enterprising publishers and benevolent or altruistic teachers have vied with each other in filling actual or alleged wants. They are not all without merits—on the contrary, some would confer honor on the literature of any country. Personal, and national, not infrequently international, experience and research are well represented in a few of them which are duly popular. They exhibit a full knowledge of European literature; and take pains in quoting from them though foreign statements or scientific results coincide with, or be in no way superior, to our own. By so doing our writers exhibit to a high degree both the honorable modesty and cosmopolitan erudition, which are characteristic of a host of American medical productions. Indeed I know in no country of any medical literature which combines to the same extent as ours practical tendencies with the persistent endeavors to fulfill the demands of scientific justice. That is why, without any carping criticism, I dare say that Germany is rather inferior in the appreciation of foreign achievements, though the decades of German medical hegemony, following the era of French predominance, have passed by to make room for modern, universal and co-operative activity.

The few German magazines published to gather from all over the world the titles of all sorts of medical output are (with almost no exception but the *Centralblätter*) storehouses which accumulate but do not digest. That is why American work is but little known in Europe, and why even the excellent German book I beg to introduce to my American colleagues, does not show the intimate knowledge of American pediatrics which has made wondrous strides during the last half century. If therein lies a shortcoming, it is not we but the German readers of the original who suffer from it. We do not look for American references in foreign books, but for the best their authors may be able to present to us—both as far as personal experience and research are concerned—and for the teaching of their best men and institutions. That is what is represented in the volume I herewith offer to the American profession.

When undertaking to edit this volume I was prepared to criticize, to reject, and to add. I have been, however, agreeably disappointed. Almost every one of the authors contributing to it is well known on this side of the Atlantic. They have all tried to furnish the best they had to give and have succeeded. If I were to select any one of the essays before us—a difficult



task—I might perhaps point to C. Keller's complete, but concise, chapters on the diseases of the new born. It is difficult to imagine anything fuller, briefer and withal more lucid than his discussions, for instance, of jaundice, pemphigus, melena, or Buhl's, and Winckel's diseases; or of the treatment of the navel, or of bathing. Still, it almost looks invidious to select any of the topics contained in this volume for special commendation.

I may be permitted to make a statement which has been suggested to me by the appearance during the last decade or two, of many of the collective volumes, and big cyclopædias, the works of many authors. With the initiative of the publishers I have no concern, nor with their—under our commercial and social conditions—natural tendency to preëempt or to share in, the profits of the market. On the part of the contributors the wish to teach additional classes of readers makes them prone to repeat what they have said in previous text-books or monographs. That seems to be natural. Others may desire to appear in good company; that is a pardonable ambition. But there are those who prefer a clinical book which is written by one man who has had ample time and opportunity to gather experience, and a broad and deep enough mind to elaborate it. To that class of critical readers I belong myself. Still, in the case of the book I here present to the American profession, I feel justified in making an exception. The articles contained in it are all written by masters of national or international renown, are evidently weighed with care and circumspection guided by learning, convey everything that is worth while possessing, are complete but brief, brief but lucid, lucid and in part eloquent, and easy reading. No extensive editing could have enhanced their value. As, moreover, the admirable translation by Dr. J. L. Salinger has provided a book at least as readable as the original, I recommend it to the attention of the professional public as a worthy and valuable addition to their libraries.

A. JACOBI.

19 EAST 47TH STREET, NEW YORK.

## LIST OF CONTRIBUTIONS

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- Diseases of the New-born in the First Days of Life.* By C. KELLER, Berlin.
- The Feeding of Children.* By AD. CZERNY, Breslau.
- The Most Common Infections of the Oral Mucous Membrane in Children.*  
By A. MONTI, Vienna.
- Acute Digestive Disturbances of Infancy.* By TH. ESCHERICH, Graz.
- Chronic Digestive Disturbances of Infancy.* By B. BENDIX, Berlin.
- Infantile Scurvy (Barlow's Disease).* By H. NEUMANN, Berlin.
- Rickets (Rachitis).* By J. ZAPPERT, Vienna.
- Infantile Scrofulosis and Tuberculosis.* By O. SOLTSMANN, Leipsic.
- Hereditary Syphilis.* By H. FINKELSTEIN, Berlin.
- Speech Disturbances of Childhood.* By H. GUTZMANN, Berlin.
- Functional Nervous Diseases of Infancy.* By H. NEUMANN, Berlin.
- Convulsions in Children.* By K. HOCHSINGER, Vienna.
- Diseases of the Nose and Pharynx Peculiar to Infancy (Excluding Tonsillitis).* By J. ZAPPERT, Vienna.
- Meningitis of Infancy, and Hydrocephalus.* By O. KOHTS, Strasburg.
- Infantile Spinal and Cerebral Paralyzes.* By A. HOFFA, Würzburg.
- Chorea Minor.* By B. BENDIX, Berlin.
- Pneumonia of Children and its Treatment.* By E. HENOCHE, Dresden.
- Rötheln, Rubella, German Measles.* By CH. BAUMLER, Freiburg.
- Measles, Morbilli, Rubeola.* By O. HEUBNER, Berlin.
- Scarlet Fever, Scarlatina.* By O. HEUBNER, Berlin.
- Diphtheria and Diphtheritic Croup.* By A. BAGINSKY, Berlin.
- Epidemic Parotitis, Mumps.* By H. FALKENHEIM, Königsberg.
- Pertussis, Whooping-cough, Tussis convulsiva.* By A. BAGINSKY, Berlin.





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# DISEASES OF THE NEW-BORN IN THE FIRST DAYS OF LIFE

By C. KELLER, BERLIN

THE pathology of the first stage of life, in its present extent, is a development largely of the nineteenth century and principally of the latter half. In this the pathologico-anatomical and embryologic investigations have had no less part than physiology and, in the last few decades, also bacteriology. Improvement in the method of investigation has furnished scientific results in each realm which have been of value also in the pathology of the new-born in that they have stimulated further research. Thus, from various points the material has been supplied with which a structure has been erected which, in regard to systematic arrangement, reveals a decided advance since the beginning of the nineteenth century. It is true that this structure is still remote from completion: Many spaces and passages lack the necessary illumination. But, thanks to the successful activity of many distinguished authors in the past century, we may be proud of the total result.

## DEFECTIVE AND PATHOLOGIC DEVELOPMENT

In the pathologic studies of the new-born those disturbances should first be considered which depend upon a *defective or pathologic development*. Here the **central nervous system** appears to be most commonly involved. That *anencephalus* or *hemicephalus*—a complete or high-graded structural defect of the brain and spinal cord—renders an extrauterine or substantive existence impossible is obvious. The most important clinical evidence which characterizes such conditions, the absence of a cover to the skull (*acrania*), makes these forms readily recognizable. *Hydrocephalus* usually reaches such a degree intrauterine that perforation of the head, therefore death of the fetus, is necessary to render the labor less serious to the mother. Exceptionally there are cases of less grade that permit the birth of a live infant either spontaneously or with the aid of instruments. Such cases show but a moderate enlargement of the skull and widening of the sutures. The subsequent fate of the infant depends upon whether the process which has caused the intracranial accumulation of fluid is arrested or whether it continues and even causes an increase in the size of the skull. In the former instance it is possible, under favorable conditions, for the central nervous

system eventually to reach a normal development, the growth of the infant in question, mentally and bodily, being such that it differs but little from others except in a slightly altered shape of the head. Naturally this possibility can arise only when the formation and development of the brain does not otherwise differ from the normal, and provided there are no sequels of the affection, as atrophy, absorption, etc. The absence of these requirements will subsequently reveal itself, provided the infant retains life, by certain symptoms of functional defect which may vary from general mental limitation up to complete idiocy, or may be of a more local nature, involving the motor and sensory spheres. But, fortunately for humanity and especially for the parents, these cases are very exceptional. The continued increase post partum of the intracranial fluid reveals itself on the one hand by the gradual but steady enlargement of the skull and widening of the sutures, and on the other by such sequels as are brought about by an increased cerebral pressure: rapid pulse, vomiting, clonic and tonic spasms, somnolence. The prognosis of the further course is exceedingly unfavorable. As a rule the infant soon succumbs. Puncture and evacuation of the intracranial fluid, so often practised in these conditions, usually has but a transitorily favorable result; the immediate improvement is soon succeeded by an aggravation. The removal of the fluid has merely a symptomatic influence and does not affect the cause of the process. Only when this is accidentally arrested can permanent relief be expected. Nevertheless, in such cases the physician is compelled to utilize this aid if only for the reason that everything possible must be done to maintain the life of the infant.

A third important clinical malformation in this sphere is the occurrence of tumor-like protrusions of the brain and spinal cord, the so-called *cerebral and spinal hernias*. The latter, which is the more common of the two, is usually situated in the median line, over the sacrum or at the lower part of the lumbar vertebral column (*spina bifida*, *myelomeningocele lumbrosacralis*). This usually presents a cystic tumor, rarely larger than a walnut, flaccid or tense, pointed or flattened, the interior of which communicates with the dilated central canal. Externally the tumor is covered by the skin, which, if very tense, is more or less thinned, sometimes having a cicatrized appearance. Under the skin is the dura. Of the substance of the spinal cord either there is none at all in the wall of the sac, or the lower end of the somewhat elongated spinal cord is inserted at the inner surface, or separate strands are found at the base of the tumor which run along the periphery of the sac. At the area of insertion the vertebral arches and the spinous processes are defective. Occasionally the sac is already opened at birth and has discharged its contents (compression during labor, especially after operative interference). Exceptionally the sac-like appendage may be absent. The skin terminates peripherally with a tumor-like thickening, centrally the tissue shows a funnel-like deepening to the point of entrance in the central canal. Much more rare are similar tumor formations in the region of the skull. Here, as a rule, in contrast to the spinal cord hernias, the sub-



stance of the brain is greatly involved. Most often there is a direct protrusion of the dropsical cerebral ventricle (*hydrocephalocele*), the cyst wall being composed of the scalp, the cerebral membranes, and the brain substance. Much less common is a cystic protrusion of the meninges alone (*meningocele*), or without accumulation of fluid, consisting merely of cerebral substance and the meninges (*encephalocele*). The points of predilection for all of these forms are the tabular portion of the occipital bone and the lower part of the frontal suture, but they have also been noted in the vicinity of the great fontanelle, the sutura squamosa, the fissura orbitalis and at the base of the skull.

The *prognosis* of hernias of the cerebral and spinal cord structures depends primarily upon the extent of normal development of the central nervous system, that is, in how far deleterious influences resulting from malformative processes (primary hydrocephalus, etc.) have left their effect upon the substance of the brain in the form of atrophy and absorption. As both of these factors are usually remote from direct inspection and the structure of the skull and the bony canal of the spinal cord in the main appear normal, their detection must depend entirely upon clinical observation. These tumors may develop further post partum, just as in hydrocephalus, or may retain their original size. In the former case there is danger of rupture by direct thinning of the skin from a nutritive disturbance, such as gangrene. But the condition is serious even if the size of the tumor remains stationary. The pressure of the infantile body or the manipulations which are necessary in the care of the child readily lead to trauma with the possibility of an infectious inflammation, which is always exceedingly dangerous on account of the vicinity of the cerebral and spinal cord membranes and of the direct communication with the ventricular system.

In an otherwise normal and well-developed child it would seem that an operative removal of the tumor, or, in the case of spina bifida, the protective covering of the cutaneous defect, should meet all of the requirements of the case. Such attempts have been reported from pre-antiseptic times. The permanent results, however, are not particularly brilliant even to-day when any active infection can almost certainly be prevented and although the wound of the pedicle heals by first intention. In most of the cases hydrocephalus arises and with the symptoms of this malady the infant perishes. The majority of cerebral and spinal cord hernias are hydrocephaloid and the operative removal of the tumor or the union of fresh edges of skin acts only in a systematic manner and is ineffectual as regards the underlying process. There is some hope of success, however, in the simple and stationary meningoceles, which, as already stated, are the most common forms of spinal cord hernias. In any event, if the tumor shows any pedicle and does not present conditions too unfavorable for surgical interference, such as a cutaneous covering for the pedicle, we should operate, if merely to prevent injury in the necessary care of the child. By a previous ligation of

the pedicle and subsequent careful cleansing of the skin the removal of the mass may usually be accomplished without opening the canal of the pedicle. In a case of mine of spinal cord hernia operated upon in this manner, the wound healed without reaction by primary intention, but in the course of ten or fourteen days the development of a hydrocephalus was apparent, to which the infant succumbed ten days later. If operative interference is impossible on account of the broad-based insertion of the tumor, nothing remains but to apply an antiseptic protective bandage to prevent injury and infection.

The deformities of the central nervous system which have been mentioned are clinically the most important on account of their frequency and their possible relief by treatment. Pathologico-anatomically they represent but few links in the chain, which covers all the gradations from complete aplasia to those deviations which may be considered but trifling abnormalities. It would lead us too far afield to detail all of these. Not only do the major deformities and their sequels possess many points of resemblance, but the same methods of treatment also prevail. These considerations will guide us when investigating the other organs of the body, to which we shall now proceed.

Within the **digestive tract** malformations are most frequently noted in the *mouth*, the *oral cavity*, and the *anus*. The first deformities to be considered are congenital clefts, such as those of the lips, known as *hare-lip*, and of the hard and soft palates, known as *palatogenesis* and *cleft palate*. The exact anatomical structure of these individual deformities is so well known through embryology and surgery that an accurate description is unnecessary and we may proceed at once to their clinical description. Of greatest importance after birth is a hindrance in proper feeding. In hare-lip this is especially marked when the cleft reaches to the nasal opening, the two presenting a combined opening in which the bony alveolar arch is usually involved, the lips being more or less shortened and correspondingly limited in their movement. In such cases the firm closure of the lips around the nipple, which is essential for the withdrawal of milk, is inhibited or entirely impossible, and artificial nourishment by the bottle is necessary. This state of affairs is still more complicated when the soft and hard palates are involved in the cleft formation. The free communication of the nose with the cavity of the mouth decreases, even to a greater extent, the efficacy of suction. Milk introduced into the mouth regurgitates through the nose. From the decomposition of particles which remain in the nose and in the mouth inflammatory processes arise (stomatitis, aphthae, etc.). These deleterious agents eventually pass with the food into the stomach and intestines, or into the larynx and the lungs by means of the inspired air, giving rise to more or less severe catarrhal processes. In addition the inhibited naso-pharyngeal respiration results in less warming of the inspired air and its contamination with dust and other admixtures, which reach the larynx. Breast-feeding is sometimes possible in complete unilateral cleft formation, provided



the cleft is narrow and the aplasia of the lips not too decided, and also provided the mother or wet-nurse has a well-developed nipple and a profuse secretion of milk. Similar conditions are necessary in bottle-feeding: the opening in the rubber nipple must not be too small and the nipple itself should be of a size to be conveniently introduced into the mouth without causing retching and nausea. With a broad unilateral, bilateral, or complete cleft formation even these measures are without avail. Feeding by means of a spoon or a cup is then necessary, care always being observed that the food is carried immediately to the base of the tongue. Prophylactically, to prevent decomposing processes, the mouth must be cleansed by a diluted boric acid solution. The relief of the deformity can be accomplished only through surgery, by the plastic union of the separated layers of tissue. In addition to the possibility of these life-threatening complications it is necessary to consider the external deformity and later the disturbance in speech. An early operation, in the first few weeks after birth, is regarded as the correct procedure. At this age the child is most apt to be quiet, cries less, and sleeps much, and the food requirement is still limited. Only in the severe cases with marked prominence of the intermaxillary process do some authors advise a postponement until the second year on account of the great loss of blood. Severe catarrh of the intestines and of the lungs is of course a contraindication; mild disturbances are relieved after a successful operation. The previous relief of infectious inflammations of the mucous membrane of the mouth and of the nose is important. The practical application of this operative method is beyond the activity of the ordinary family physician and belongs to the realm of the specialist; it will therefore not be considered in detail at this point. In this connection reference may be made to text-books on surgery.

The most important *malformation of the anus* is *atresia congenita*, due to the non-union of the terminal bowel which has been invaginated from without with the lower portion of the intestinal canal. In the majority of the cases the terminal gut is absent and in place of the usual anal opening there is only a moderate retraction of the skin. More rarely the entrance of the terminal bowel exists but has a blind ending below the intestinal canal, from which it is separated by a layer of tissue. Roser has called attention to the occurrence of simple epithelial agglutination at various heights. This deformity is recognized by simple inspection or through the non-discharge of meconium. The principal danger lies in the decomposition of the contents of the intestinal canal by the bacteria which enter with the introduction of air and later of food. Just as in obstruction of the bowel from other causes, a septic peritonitis may finally develop. Therapeutically the only relief is through operation, which should be performed as early as possible on account of the threatened calamity.

When the inhibition in development has occurred earlier, at the time of the *formation of the cloaca*, the conditions are even more complicated. Those forms then develop in which the intestinal canal terminates in the

bladder or urethra (*anus vesicalis* or *anus urethralis*). Such infants perish soon after birth in consequence of ichorous inflammations of the bladder, of the urethra, and of the kidneys. The operative production of a condition resembling the norm must be attempted as early as possible, but, as is evident, is extremely difficult.

Of other persistent deformities *stenosis of the cardia*, *constriction*, *obliteration* or *complete interruption in the course of the remaining intestinal canal* must be mentioned. The last condition is usually associated with the symptoms of adhesive peritonitis, but whether this is the cause or the consequence of the embryonic disturbance is uncertain. To me the former appears to be much more probable. Here the deleterious consequence of the intake of food and its digestion—the putrid decomposition of the stagnating intestinal contents—is all the more serious because at first the diagnosis is impossible and the condition may not be suspected until after the absolute failure of all therapeutic measures and only *by exclusion*. By this time, as a rule, the child is so debilitated that even if operation were possible it would be entirely useless. The *prognosis* of these conditions therefore is altogether unfavorable.

Within the **urinary apparatus** the conditions of most vital importance are *defective formations of the kidneys*, which give rise to insufficiency. The most familiar of these is *congenital cystic kidney* which is usually bilateral and consists of a small cystic degeneration. Large tumor formation, with great distention of the abdomen, leads to trouble during delivery, causing the death of the child either directly or from the necessary artificial aid. Even if born alive the child usually perishes within a short time. As a rule a positive diagnosis can be made only by autopsy. Bilateral localization by palpation of a tumor in the upper segment of the abdomen would suggest the condition. High-graded *hypoplasia* or *complete aplasia* of both kidneys is only a part phenomenon of a condition present also in other organs and necessarily is incompatible with life. The unilateral presence of this inhibited embryologic development is commonly compensated by a corresponding hypertrophy of the other kidney and thus all damage to the development of the individual is prevented. Accordingly the post mortem evidence of unilateral hypoplasia or aplasia in an adult is one of those interesting surprises which were unsuspected during life.

In addition to these direct malformations there are many which have a secondary, indirect effect upon the kidney by hindering the off-flow of urine. *Malformations of the ureter* (obliteration, high-graded constriction, kinking, valvular closure) result in complete or incomplete *hydronephrosis* of the side involved. If the obstruction is more *distally situated*, *within the ureter* (complete closure, constriction, stenosis of the external orifice; in hypospadias or epispadias or with an otherwise normal development, phimosis) the bladder will be affected first (over-filling, over-distension) and in the process of backward stasis both kidneys will be involved. Whether these sequelæ will develop intrauterine depends upon the magnitude of the



obstruction. Complete closure must have its effect even intrauterine. On the other hand deformities of lesser degree may cause a disproportion of the lumen and only gradually appear in extrauterine life under the influence of the urinary secretion which steadily increases with the ingestion of food. The *prognosis* depends upon the degree of the renal disease and is, therefore, decidedly more grave in the intrauterine forms and with the involvement of both kidneys. It is most unfavorable in those very exceptional cases in which there are also other malformations and when there is complete occlusion of the urethra. It is the duty of the obstetrician to inspect the channels of outlet at once post partum for an immediate recognition of these conditions. The discharge of urine during subsequent days must also be noted. In the more marked forms there is also a prominence of the lower abdominal segment, due to the distended urinary bladder or to the presence of a more or less distinct renal tumor. It is scarcely necessary to mention that in the conditions of complete closure of the lower urinary passages which in the course of time produce a degeneration of the renal tissue nothing is to be expected from therapy. Such *treatment* is futile, as has already been stated, on account of malformations in other vital organs. But even in unilateral hydronephrosis an expectant treatment at first is indicated. For some time the tumor remains stationary and of moderate size. The newly born infant is not in a condition to bear the effects of a serious operation and quickly succumbs to shock. An effective treatment is only possible with those isolated constrictions at or near the surface. This consists of a surgical dilatation. The time for operation depends upon the degree of the deformity. For the method of operation recourse can be had to the text-books on surgery.

In addition to the deformities of the urinary organs already mentioned there are many others: *dystopia (malposition) of one or both kidneys, horse-shoe kidney, duplication and abnormal outlet of the ureter, hypospadia, epispadia, ectopia vesicae*, etc. In the first few weeks of life these are of less serious import as they have no influence upon the existence and the further development of the individual. Some of these conditions have merely a scientific value at any age or are only of importance in a differentio-diagnostic respect. In hypospadia and epispadia of males sexual impotence in later years may require the relief of the malformation provided that technically such an operation is possible. Congenital abdominal fissure communicating with the bladder, in which the prolapsed mucous membrane of the bladder is exposed to injury and infection and the adjacent epidermis can not be protected from eczematous affections on account of the constant contamination with urine, is not operated upon as a rule before the fourth year or even later.

Important defects in the development of the **respiratory tract** occur only when there is marked malformation of other organs. A disturbance of the respiratory function is brought about indirectly by *incomplete closure of the thoracic cavity—diaphragm and thoracic wall*. Congenital cleft of the

diaphragm prevents inspiratory distension of the lungs; with the corresponding muscular action the adjacent abdominal organs penetrate the thorax and the child perishes at once *post partum*. A similar insufficiency results from the various forms of thoracic cleft, the more marked of which, as is well known, are associated with complete *ectopia cordis*. In the latter the cause of death may be determined by simple inspection; the former developmental deformity can naturally be determined only by autopsy.

Within the **circulatory apparatus** the degree of development of the *heart* is of the greatest importance. Complete absence or a functionless rudiment is found only in twins from one ovum. The heart of the normal twin simultaneously supplies the circulation in the *acardiacus* (*omphalosite*). The two may be intimately adherent (parasitic) or may be separate (free *acardii*). In the latter form the communication of the vessels is within the placenta. The life of the *acardii* ends with the separation from the host, therefore with the free *acardiacus* at birth, that is, with the tying of the umbilical cord. If the parasitic *acardii* belong to the double malformations those which are free present very marked monstrosities (*acephalus*, *acormus*, *amorphus*, etc.).

Of much greater importance than these passive rudimentary structures are the developmental defects of the self-acting heart, the so-called *congenital vitia cordis*. Their pathologico-anatomical and clinical investigation we owe largely to the labors of the latter half of the past century. According to these researches the frequency of this condition has been greatly under-estimated in former times. The severe forms with absolute incapability of life, or a marked disturbance which appears very early, are regarded as rare. On the other hand there are a comparatively large number which do not show any or but very slight symptoms until a greater corporeal exertion (for example, the first attempts to walk) increases the activity of the heart.

Developmental errors may appear at any stage of the formative period of the heart. The earlier their occurrence the greater are the consequences, in that a primary deviation results in further defects of development. With each advance in the development the circle of this direct influence is narrowed so that, finally, when the heart has reached the stage of intrauterine or fetal completion single anomalies are very seldom observed.

In the *combined or cumulative forms* the large arterial vessels (aorta, pulmonary artery) are commonly involved. A division of the truncus arteriosus communis either has not taken place at all or is rudimentary. This has occurred at the cost of one of the vessels (atresia or stenosis of the pulmonary artery or aorta), or the intervacular wall has taken an abnormal course and has connected the pulmonary artery with the left ventricle, the aorta with the right ventricle, or both vessels with one ventricle, usually the right. Simultaneously, especially with a hindrance in the off-flow through the arterial orifices, there is a defect in the formation of the septum atriorum or septum ventriculorum. In addition, atresia and stenosis may result in a patulous or even a more marked development of the ductus



Botalli in order to aid the deficiency in circulation which would otherwise arise distally. Finally, owing to the difference in the amount of work performed because of the abnormal distribution of blood in the heart, one of the ventricles may develop beyond the normal extent while the other is more or less retarded. It is apparent that in this manner malformations may arise which are not equal to the increased requirements at the completion of birth, that is, to the change from the fetal circulation. Such infants perish during birth or soon afterward from *cardiac syncope* resembling profound asphyxia. Another group survive the first attack but rapidly present the significant symptoms of a disturbed cardiac activity. The most familiar of these symptoms is *blueness of the skin or congenital cyanosis*. Naturally, this symptom only appears in those cases of developmental defect in which there is a further backward flow of the blood into the veins of the body. At first, and even later, the regions most markedly involved are the face, the ears, the hands, and the feet. The lips, the mucous membrane of the mouth, and the conjunctivæ may also be distinctly altered. All shades are observed, from the deep blue which is conspicuous even to the laity to a bluish discoloration of the mucous membrane scarcely noticeable to the expert. Crying, vomiting, cough, bodily movements, increase this symptom just as complete rest ameliorates the condition. The hands and feet are cool to the touch. Should the infant live, club-like swellings develop on the nail phalanges (*drum-stick fingers*). Congenital cyanosis is an infallible sign of a congenital anomaly of the heart. There are, however, congenital defects of development in which this symptom is absent, i. e., when the condition of backward flow into the veins, mentioned above, is not present. The complexion then presents a conspicuously pallid or grayish yellow appearance which in later years may readily be mistaken for simple anemia or chlorosis.

Infants suffering from congenital defects of the heart show peculiarities in their behaviour and habits. They are more quiet and sleep much. The voice is hoarse and without power. The movements are rather sluggish and phlegmatic, lacking the ordinary activity. Nursing from the breast or bottle is very slow, with long pauses as though the child were exhausted and suffered from lack of air. In addition to these minor consequences of decreased absorption of oxygen, due to a stasis of blood in the lesser circulation, there are sometimes attacks of dyspnea, at first perhaps associated only with crying, vomiting, cough, etc.; later these attacks occur at short intervals, without special cause, and are augmented to actual asthmatic attacks. If we add to this the tendency to epistaxis, hemoptysis, catarrh of the respiratory organs, and a lessened resistance to other diseases, especially those of the gastro-intestinal tract, we may readily realize how unfavorable is the prognosis. The greater number of infants with congenital disease of the heart succumb within the first twelve months, and but a very small number reach the fifteenth year. Very few survive to an adult age, even to the fortieth year or more. Death results either directly from compensatory

disturbance (dyspnea, dropsy) or from complications, among which gastro-enteritis in the first year of life and tuberculosis in later years play the most important part.

For the clinical diagnosis the results of auscultation as well as congenital cyanosis are important. The demonstration of cardiac murmurs soon after birth is sufficient. Functional and hemic murmurs do not yet arise. The appearance of an acute disease of the heart immediately after birth is exceedingly rare and is then associated with corresponding constitutional symptoms. The differential diagnosis of acquired valvular lesion does not come into question until later. Among the important general differential factors we find that the murmur of congenital disease is almost always systolic in time and most distinct at the base of the heart, not at the apex, that the definite auscultatory signs are in marked contrast to the signs upon percussion, which are slight or absent, and finally, that the apex beat (the impulse) is never increased but rather weakened. The presence of other malformations is of some value in establishing a congenital predisposition. To enter upon a more explicit description of special deformities and their explanation would lead us too far. Such descriptions will be found in the works of Assmus, Rauchfuss, Freymann, Lebert, Hochsinger, etc. The minute analysis of these combined congenital valvular lesions sometimes offers great difficulty. However, the many confirmations of the earlier diagnosis by the necropsy findings prove sufficiently that this object can be met by careful study, at least so far as the grosser lesions are concerned.

In regard to treatment, those measures must first be considered which are likely to improve the general condition, therefore proper food, sufficient warmth. The great danger of intercurrent disease of the intestines and of the respiratory organs requires particular care in the preparation of the artificial food and of the necessary utensils (bottles, nipples, etc.), and a careful airing of the room. When compensatory disturbances arise minute doses of digitalis and strophanthus are of service. Naturally the improvement thus produced is only transitory; a permanent, even though partial, restitution is beyond all hope.

In contrast to these combined congenital cardiac lesions the *individual valvular defects* are considered quite rare. Many of them escape observation as they give rise to no particular symptoms. The remainder are noted when increased effort is required on the part of the circulation (corporeal exertion, disease of the respiratory organs, etc.). As the heart has otherwise reached a substantive normal development it is able to a certain extent to overcome the slight disturbances due to a single congenital lesion. The most common of these are: *persistence of the foramen ovale*, *patulous ductus Botalli*, *slight defect of the membranous portion of the septum ventriculorum*, and finally, changes in the *auriculo-ventricular openings*, particularly of the *tricuspid*. The general characteristics coincide with those of the combined developmental defects already described. In regard to the special symptoms reference must be made to text-books on internal medicine. The prognosis,



naturally, is much more favorable. Treatment is only necessary when compensation fails and is then in accordance with general principles.

**Congenital rupture of the umbilical cord—hernia funiculi umbilicalis**—must be regarded as a serious defect of development. While under normal circumstances the abdominal cavity is closed in the umbilical region by the growth of the abdominal walls to a narrow fibrous ring which permits the vasa umbilicalia alone to penetrate, there is at times a broad or a narrow cleft by which the intestines enter into the lower portion of the umbilical cord. Under such circumstances a flaccid, broad, vesicular or pointed tumor of the umbilical region is presented, the cord being inserted upon its opposite side. According to the size of the tumor and the width of the abdominal cleft only portions of the intestines are protruded or also parts of the liver, of the stomach, and of the spleen. In the extreme cases the mass without the abdominal cavity may preponderate to such an extent as to fully justify the term *eventration*. These extreme grades are commonly associated with other marked malformations which render subsequent existence impossible; some of them intrauterine give rise to difficult labor which leads directly or indirectly to the death of the infant in consequence of the necessary aid during delivery. After birth the danger of rupture of the umbilical cord lies in the composition of the walls of the sac. This consists only of amnion and peritoneum. The margin of skin is usually at the base of the tumor but sometimes even this has not been reached; more rarely the skin extends for a little distance over the tumor. The amnion, like the umbilical cord, undergoes necrobiosis *post partum* and if there be a contamination gangrene may appear. A further consequence is the development of purulent peritonitis, which soon results in death. The only radical cure is by operation: a round incision corresponding to the cutaneous margin, reposition of the abdominal viscera, and a union of the abdominal wall by suture as in laparotomy. This operation may be performed early without opening the peritoneum provided the amnion does not adhere to it too firmly and there are no adhesions of the intestines. The facility of the operation, as well as the prognosis, depends primarily upon the size of the tumor or the width of the cleft. If the latter is so great that the skin cannot be united at all or only with extreme tension it has been proposed to utilize the amnion as a cover. This, however, is of little benefit. It is better to relieve the tension by loosening the layers of the abdominal wall with lateral incisions. Such serious operations, even in well-developed infants, are never of especially favorable prognosis. They perish soon afterwards from shock, even without the symptoms of infection. In the pedicular hernial sacs, after reposition of the content the pedicle is tied. Aside from the fact that the intestinal wall may be included in this the predisposition to umbilical hernia still remains. These obstacles can be overcome only by operative incision and suture. If the relatives refuse operation nothing can be done but to apply an antiseptic protective bandage, which prevents infection on the one hand, and on the other prevents the prolapse of the intestines

in consequence of the abdominal press. In the hernias of medium size these measures will scarcely prove effectual for any length of time. In the smaller hernias a gradual growth of skin is sometimes noted. For the thorough closure of a narrow cleft the use of paraffin injection is necessary—a method which has been found valuable in congenital umbilical hernia.

In addition to the varieties with distinct tumor formation the less prominent forms are of great importance, especially as they readily escape the observation of the physician. The lower part of the umbilical cord and its insertion into the abdominal wall may merely be abnormally broadened. The intestine lies mostly within the abdominal wall or protrudes but little beyond. Very often there is a Meckel's diverticulum. In tying the umbilical cord at the level of the abdominal skin the wall of the intestine may easily be included in the ligature. With the falling off of the cord there is an *anus praeternaturalis*. If the ligature is applied at a distance from the point of insertion the dropping off of the cord may leave an opening in the peritoneum into which the uncovered intestine may penetrate. The immediate consequence is purulent peritonitis.

In the descent of the ladder of these developmental defects those cases yet remain in which there is no actual *hernia funiculi umbilicalis* but the umbilical ring is merely broader than normal. In these cases an *umbilical hernia* develops soon after birth (therefore acquired) which, in contrast to the congenital navel hernias, usually protrudes above the umbilical cicatrix. In all of these lesser varieties the fundamental principle of prophylaxis and treatment depends upon an early recognition of the condition. This is greatly aided if the rule is always followed not to tie the umbilical cord until the child actually cries. The intestines are then forced forward and smaller clefts are more easily noted. Otherwise the treatment is identical with that of larger hernias and consists in operation, the prognosis of which is decidedly more favorable on account of the slighter intrusion. To prevent a later umbilical hernia the navel bandage should be kept on the child for three months and the pressure in the umbilical region increased by layers of cotton under the bandage. For an umbilical hernia that has already developed the use of an adhesive plaster bandage with a pad included has been recommended. Good results have been recently attained by peritoneal injections of paraffin by which a radical cure appears possible. An attempt should be made to ascertain whether these injections would not be equally as efficacious if given subcutaneously or subperitoneally, as they are submucously in incontinence of urine.

Finally, **malformations of the extremities** must be described. Only those are of practical interest in which treatment is able to bring about improvement. Among them are the *rudiments of superfluous fingers and toes, web formation, syndactylia, club-foot, club-hand, congenital dislocation of the femur*. Although, in the last decades, in the endeavors to better the permanent results the necessary operation or correction has been attempted as early as possible, it is the opinion of all authors that the first few weeks

are not suitable. In regard to the special operation and the time of its performance recourse must be had to surgical text-books.

Thus far we have considered only the defects of special organs or systems, but *the entire infantile structure at birth may present a lack of development* or a deficient vitality. This condition, designated **general debility**, is most commonly the result of a premature interruption of the pregnancy, of which the most varied diseases of the mother or physical or psychical trauma may be the direct cause. Infants born at term are debilitated only as the result of a twin labor, both infants being affected or one alone, the other being quite strong and normal.

Debilitated infants born at full term are exceptional. Deficient nourishment of the fetus is the usual cause and is due to an extremely unfavorable condition of living or a chronic disease of the mother, the deleterious influence not being so serious as to cause a direct interruption of the pregnancy. Children under 2000 grams in weight and measuring less than 42 cm. are regarded as debilitated. The color of the skin, at first of a bright, glistening red, gradually becomes pale with a bluish tinge and cyanotic. The deficient development of the subcutaneous fatty tissue is shown by a marked wrinkling of the skin, which lends to the face a wizened appearance. The dorsum and the extensor surfaces of the extremities show a thick growth of woolly hair. The sutures and fontanelles are wide, the nails incompletely developed and soft, not reaching to the tips of the fingers. The insertion of the umbilical cord is below the middle, between the symphysis and the ensiform process. In the genital organs of boys the *descensus testiculorum* is lacking, and in girls the clitoris and smaller labia protrude considerably from the but slightly prominent labia majora. The most important differences from normally-born children are the weak pulse, at first rapid, but later slowed, the superficial respiration (whimpering rather than crying), the subnormal temperature, and the inability to suckle, i. e., to withdraw a sufficient quantity of milk from the breast. Children weighing but 1000 grams usually perish the first or second day after birth, and in spite of all care those of 1200 to 1500 grams rarely live. The principal damage is to be found in the deficient expansion of the lungs, which results in softness of the ribs and a relaxation of the walls of the thorax. Upon every attempt at inspiration the anterior thoracic wall is depressed by the external pressure of air. The limited circulation due to this is shown by marked cyanosis of the skin. The gradual decline of the functional activities, which were extremely weak from the beginning, terminates in death in the first or second week. A child weighing 1500 grams or more may live with a certain amount of care. For the *prognosis*, in addition to the weight the temperature of the body is important. The lower the rectal temperature the more serious the outcome. Infants who never show a higher temperature than 35° C. (95° F.) do not live. The prognosis is more favorable in proportion as the respiration and circulation improve and the food is ingested. This reveals itself primarily by an increase in temperature and later by a gain in weight. Infants who



fulfill these conditions in the third week *post partum* present in general a more favorable prognosis, but it usually requires from six to eight weeks for the minus of the congenital debility to be adjusted. Many infants remain delicate for years, some of them for life.

In addition to this danger of weak vitality debilitated infants present less resistance and are more susceptible to infectious diseases. This condition may be evident in the process of healing of the umbilical cord. Putrid decomposition, septic infection of the umbilical wound and thence to the rest of the body, is particularly common. There is a decided tendency to the development of aphthæ upon the mucous membrane of the mouth and pharynx and their further distribution to the esophagus, larynx and intestinal canal. The deficient activity of the lungs further predisposes to the development of bronchitis and broncho-pneumonia. Finally, the enfeebled circulation often leads to the development of sclerema, which distributes itself over large areas of the body with a further decrease in the temperature and a slowing of the pulse.

In the *treatment* of debilitated infants it is necessary first to consider their ready susceptibility to low temperatures. Directly after birth they are to be bathed once in water at a temperature of 36-37° C. Afterward merely simple washings with warm water should be given, exposing only the part to be bathed. The same fundamental law of the slightest cooling and the greatest possible retention of heat should be observed in regard to the clothing. Cotton or flannel is best and should be heated before each change. The crib should be kept at a uniform temperature of 32-34° C. by hot bottles placed alongside and beneath the child. To prevent burns the bottles should be wrapped and the clothing next to the child must not be too hot. The necessary control is obtained by thermometers placed at the side and under the infant. The inspired air should be of the proper warmth and the child be not too greatly cooled during dressing, bathing, etc., and the temperature of the room should be uniform, between 22° and 24° C. To avoid cooling of the air by opening the doors and windows for the purpose of ventilation there should be two adjoining rooms in which the child can be placed alternately while the other room is aired. The most uniform and best warmth for the child is obtained near the stove. In place of the hot water bottles thermophor pillows and plates have recently been introduced into commerce which retain heat for a long time.

This method, which is of the simplest, has the advantage that on account of the low cost it may be employed by persons possessing limited means. It is true the fundamental condition of constant warming of everything about the child, particularly of the inspired air, is very incompletely fulfilled. That, however, by this means a positive result may be obtained has been taught me by a case from my out-patient practice, in which, with very limited means, it was possible to maintain a child born between the sixth and seventh months and weighing not quite 800 grams. An accurate application of this simple method undoubtedly requires a great amount of trouble



and care. Although by careful observation and the aid of a thermometer it may be determined when a change of the heating apparatus becomes necessary, the constant day and night renewal demands much watching on the part of the nurse. Far easier is the use of a bath-tub with double walls which may be filled with warm water (Credé). Every three or four hours the water must be renewed. Less serviceable, in practice at least, is the employment of a permanent water-bath (Winckel) and the object of heat at all sides as well as of the inspired air is no better fulfilled by this method. This can only be accomplished by incubators, of which quite a number have been constructed and recommended in the course of the last few years: The earlier apparatus of this kind (Tarnier, Hearson, Odile, Martin) was very complicated and expensive but a decided simplification has recently been attained and their use particularly in well-to-do practice can no longer be objectionable. The self-acting heat regulators are a great improvement and more certain. There is no place in this article for a minute description of the various models and their utility. For this I must refer to the excellent reports of Finkelstein, Monti, Rommel and Tolano. It must, however, be remarked that the value of a closed heating apparatus is not only founded on the result of a constant temperature, but that a sufficient quantity of pure, unused, and properly moist air must be conducted to the child. For this reason direct heating of the introduced air by a gas, spirit or petroleum flame is absolutely unsuitable since we are then dealing with combustion or exhausted air, the quality of which cannot then be improved even by the proportionate amount of moisture it contains. The only incubator I shall mention is the older Auward apparatus with which I have had satisfactory results. The very tedious and awkward method of heating through cases of water is greatly simplified, since the introduction of electricity into houses, by the substitution of electric globes.

Of just as great influence as the retention of heat (to retain the life of debilitated children) is the food. The best food unquestionably is mother's milk. Children who cannot nurse or do not nurse sufficiently should have the milk given to them with a teaspoon. Milk withdrawn by the milk-pump from the breast of the mother or wet-nurse and given to the child by a tube is not advisable because the necessary cleanliness cannot be observed. If the child cannot swallow, nourishment by the stomach tube (Nélaton's catheter) is necessary. The milk must be administered to the child every three hours day and night in quantities of 20 to 40 grams. With children who are able to suckle it is absolutely necessary to control the amount of food by weighing before and after the feeding. Should the quantity prove too small the lacking quantity must be given with a spoon. In artificial feeding it is usually best to give a combination of one part milk and two parts thin gruel with some milk sugar. Some authors employ Gärtner's, Backhaus's, or Voltmer's mixture. Monti and Passini advise one part milk with two parts whey.

• Finally, the pulmonary activity of the infant requires attention. Prophyl-

lactically a frequent change of posture prevents the development of hypostasis. Washing of the body with warm water morning and evening with slight friction of the skin, together with crying, leads to deeper inspirations which decidedly facilitate circulation. If the respiratory movements are insufficient, raising of the trunk below the shoulders and closing the nasal opening a few times during the day are advantageous. Naturally, all of these measures must be undertaken with careful avoidance of cooling of the infantile body.

## INFECTIOUS DISEASES

We now come to the infectious diseases which may be transmitted from the mother or the father to the fetus. Of these the best known is *syphilis*. The conditions of its transmission and the nature of the symptoms have been described in another article<sup>1</sup>. In regard to *tuberculosis*, up to within a short time the view was accepted of a hereditary predisposition which not rarely revealed itself externally by a feeble physical development. With advanced tuberculosis of the father the secretion of sperm containing bacteria may at this time be regarded as proven. There is no pathologico-anatomical proof, however, of an actual transmission to the fetus in this early stage of development. In regard to the possibility of infection on the part of the mother the conditions are different. Absolutely positive cases of maternal transmission of tuberculosis have been reported in the last few years by Rindfleisch, Damm, Birch-Hirschfeld, Lehmann, Schmorl and Kochel, Anché and Chambrelent, Bar and Renon. I mention only those authors who were able to demonstrate both pathologico-anatomically and bacteriologically an actual tuberculosis in the fetus as well as in the mother. In the fetus recent as well as older caseous or even calcified tubercle foci were found, the specific composition of which could be demonstrated not only microscopically but also by culture, by the demonstration of bacilli, and the pathogenicity has sometimes even been strengthened by inoculation experiments. The transmission by the placenta, as well as the nature and the localization of the specific disease in this organ, also forms the subject of these highly interesting investigations. In some of the cases the disease in the mother was acute miliary tuberculosis, in which the complete infiltration of the body, therefore a transmission to the placenta and thence to the fetus, appears obvious. In addition to this affection, which in the totality of tuberculous diseases is quite rare, is the positive finding of tuberculous foci in the placenta in the chronic varieties of tuberculous pulmonary phthisis, through which the possibility of a maternal transmission to the child attains a certain importance. In regard to the age of the infant, in a portion of the cases the condition was found in the fetus at the second half of pregnancy, the mother having died in the agonal stage of Caesarian section, or the pregnancy having been for some reason spontaneously interrupted. The fetus was still-

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<sup>1</sup> See article on "Hereditary Syphilis."

born or soon perished post partum. In a second variety of cases the infant lived for some days or weeks (death between the end of the first and the fourth weeks). The tuberculous focus, corresponding to the intrauterine circulation, occurs principally in the liver and spleen. Disease of the lungs is much less frequent. In addition to these vascular forms there are cases of lymphatic distribution, characterized by retroperitoneal, mesenteric, bronchial and portal lymph-gland tumors. The maternal transmission in the severe acute and chronic forms of tuberculosis may therefore be considered proven. In regard to the great rarity, however, with which tuberculous disease is observed in the first years of life, this result must be regarded as exceptional. For the majority of cases, as regards the influence of tuberculosis in the mother, we are dependent upon the view of a hereditary predisposition in the sense of a more ready susceptibility to external infection by means of milk, air, substances contaminated with sputum, etc.

In regard to the acute infections, such as *variola*, *measles*, and *scarlatina*, we are dependent at the present time for the recognition of the disease in the fetus upon the clinical symptoms alone. The properties of the causal virus are at this time so little characteristic bacteriologically that it cannot be utilized in diagnosis. Intrauterine transmission from the mother to the fetus has been proven most positively in *variola*. Women suffering from *variola* have given birth to premature children, and children have been observed at term with the pustules of *variola* or distinct, characteristic cicatrices of the affection. Therefore *variola* of mild form may exist in the fetus without causing an interruption of the pregnancy. The transmission and the course of the pathologic process do not occur simultaneously with those of the mother but usually somewhat later. Whether there is a special tendency to fetal infection during the stage of suppuration, as some assume, has not yet been positively determined. Nevertheless the pustules of *variola* have been found at birth upon the off-spring of women already convalescent from the disease, while mothers who were still ill of *variola* have given birth to apparently healthy children who were not affected until a few days later. The latter observation forces us to suspect the apparently healthy child and accordingly to isolate it. In a twin pregnancy one child may be infected and the other healthy. This extremely interesting circumstance seems to indicate that there is a lesion in the epithelium of the chorion (Wolff) whereby the placenta itself becomes susceptible to the virus. This coincides with the fact that in comparison to the total number of births in women suffering from *variola* the intrauterine transmission is exceptional; the premature or full-term infants show no signs of this process at birth or subsequently. Such healthy off-spring of mothers suffering or convalescent from *variola* are always immune to the disease and to vaccination. This interesting fact which was in former times only conceivable by the assumption of a *variola* running its course *in utero* without definite localization and its results, to-day becomes much more comprehensible through the influence of a fetal antitoxin formation stimulated by the maternal disease. This immu-



nity of the child resulting from the variola of the mother has stimulated attempts to protect the fetus by vaccination of the mother. Judging from present results the effect of this process appears to be very questionable, since a subsequent vaccination of the child is frequently successful. The influence of maternal vaccination, however, cannot be denied entirely as the pustules in the infant show a slower and slighter development. The best results of vaccination occurred in women who had been vaccinated in infancy; the poorest results in those whose first vaccination occurred within the last fourteen days of the pregnancy.

Most of the reports regarding intrauterine transmission to the fetus relate to *measles*. In how far the individual case may actually be considered positive depends upon the requirements for a positive diagnosis of this affection. Clinically the demonstration of a rise in temperature, catarrh, and a cutaneous eruption which fades after a few days, with small-flaked desquamation of the epidermis, is necessary. In the rudimentary forms one or another symptom may be less marked or even absent, but for a positive diagnosis a rise in temperature, even though moderate, is probably always necessary. All reports must be excluded as unreliable which mention merely a later extensive desquamation in the new-born infant, therefore, in which an exanthem was not present. Desquamation, as is well known, occurs in all new-born. Its extent and the time of its appearance is subject to such great variations even under normal circumstances that to draw conclusions from it in regard to the presence of complications is impossible. Nevertheless there are quite a number of reports of an eruption upon the skin which is occasionally described as a distinctly characteristic exanthem of measles or is designated as macular. The skin of the new-born, however, at birth is of a bright red color, which is all the more decided the stronger the respiration and the more the infant cries. In the succeeding days the discoloration gradually fades. It need scarcely be remarked, therefore, that the recognition of a cutaneous affection, the chief characteristic of which is redness, even though distributed in a definite manner, must be exceedingly difficult and readily subject to error. A positive sign is the temperature curve, but there is absolutely no mention of this in any of the reports. Rüter, who noted a rectal temperature of 40° C. (104° F.) in a new-born infant, found no eruption upon the skin but only a somewhat more decided desquamation on the fourteenth day. As in this case, the mother showed an axillary temperature of 39.5° C. (103.1° F.) a transmission of heat to the child may be assumed, particularly as the latter after twelve hours was found to be entirely free of fever. If there was no rise in temperature in the other cases then the cutaneous affection must be placed upon the same level as those which arise from the effect of drugs or in consequence of intestinal catarrh; in other words, it must be regarded as of reflex or trophoneurotic origin. The cases that have been reported are too inadequate to prove an actual occurrence of measles. For a definite decision of this question a rise in temperature at least is absolutely necessary. There are, however, reports of two cases in which, in



addition to the eruption upon the skin, the characteristic catarrh (conjunctivitis, sneezing, cough) was present. One child (Hedrich) was born with these symptoms. In the other case (Heim) the catarrh appeared upon the fourth day and the exanthem upon the eighth day, post partum. As the minimum period from incubation to the eruption of the exanthem is supposed to be thirteen days there would be no objection to considering the latter case as one of intrauterine infection. Sneezing is a well-known peculiarity of all new-born infants. The other catarrhal symptoms, however, are very suspicious, and if there are any reports at all in the literature which warrant a diagnosis of measles in the new-born the two last-named cases would have to be first considered. In these cases there is no mention of a rise of temperature. The opinion of many authors that unquestionable cases of genuine intrauterine infection of measles have not yet been reported or observed seems fully justified after the foregoing statements.

The same remark is true in regard to *scarlatina*. The positive examples of an earlier period (Meynet, Asmus, Potier, Ferrario, Pourtual, Gregory, etc.) offer too few points of support for the actual presence of this infectious disease or do not exclude the possibility of some other method of transmission. In regard to the negative results, according to which the child remains well, the confusion with a cutaneous exanthem in septic disease of the mother, which so often has been the cause of error, is a weighty argument. In recent times when the differential diagnosis of the acute exanthemata has been especially perfected, the observations of births of entirely normal children has become more prominent. Scarlatina, however, is an exceedingly rare disease in adults and the percentage of cases in pregnant women is correspondingly very low. Further, even in variola, the number of cases of intrauterine transmission is exceedingly small. If, therefore, a positive condition has not yet been noted the possibility of the occurrence of intrauterine infection can by no means be excluded. In all such instances it will be well to isolate the new-born child. There is much less danger that the infant may be infected by the mother later, as children who are well when born usually remain so and therefore in all probability have been immunized.

Of great importance in the influence of maternal infectious diseases upon the fetus are the recent researches in regard to *enteric fever* (typhoid fever). Here, just as in tuberculosis, we are dealing with a pathogenic agent which is distinctly characterized bacteriologically. Reher was the first to obtain cultures of typhoid bacilli from the fetal organs, the fetus having been born at six months, during the third week of an attack of enteric fever in the mother. This finding was soon confirmed by Neuhaus, Chantemesse and Widal. Although in these reports the characteristics of the bacilli are not so distinctive that they may be regarded as absolutely conclusive, all doubt is removed by the further investigations of Eberth, Ernst, Hildebrandt and Dürk. The first author found the pathogenic agent microscopically in smear preparations of the blood from the heart, of fluid from the spleen and liver, and in the intervillous spaces of the placenta. Ernst

showed positive preparations from the spleen, the brain, the bone-marrow, the skin, and the muscles of the abdomen and heart; Hildebrandt from the spleen, kidneys, liver, mesenteric gland, from the veins of the navel and from the blood of the heart. The bacilli were found chiefly within the lumen of the vessels, more rarely between the tissue elements. The possibility of a post mortem overflowing which might have happened formerly with the simple culture index is removed by this microscopic intravascular finding and proves that the transmission to the fetus positively took place during life. Pathologico-anatomically every other characteristic local lesion of enteric fever, especially in the intestines, was absent in the fetus so that the disease must be regarded as a pure general infection, or, as Reher suspected, *blood typhoid*. The transmission to the fetus in enteric fever after the outbreak of the maternal disease is apparently somewhat delayed; the cases that have been investigated occurred prior to the end of the second week of the affection. In how far the intrauterine infection is responsible for the death of the fetus in the cases that are still-born is very difficult to decide. In the course of enteric fever in the mother there are many complications by which the vital force of the fetus may be destroyed, or so far lessened, that the deleterious influence of the bacteria needs comparatively slight assistance. To this category probably belong those cases in which the absence of any reactive change in the infantile body lends to the process of infection rather the appearance of a simple overflowing with the toxins of the disease. More suspicious are the cases in which there was a distinct parenchymatous inflammation of the liver and spleen in the fetus. Of most importance and interest, however, are those conditions in which the child was born alive and succumbed later, apparently to the consequences of the infection. A case of this kind was reported by Ernst: A child born between the thirty-sixth and thirty-seventh weeks of pregnancy died suddenly upon the fourth day from some undetermined cause with peculiar symptoms (jaundice, eruption). At the autopsy no special disease was discovered but bacteriologically there was found a flooding of the entire body with Eberth-Koch-Gaffsky bacilli. In opposition to those positive bacillary findings there are quite a number with negative results (Simmonds, Fränkel, Kinderlen, Birch-Hirschfeld). This proves that the intrauterine transmission is by no means the rule; on the contrary, it appears to depend in part upon the local changes in the placenta, in part upon the particular severity of the affection in the mother, and therefore, upon the increase of bacilli in the intervillous spaces of the placenta, and perhaps also upon other requirements at present unknown. Recently there have been reports regarding the Gruber-Widal reaction in the fetus; the number of positive and of negative results are about equal. The observations are highly interesting in which the bacteriologic investigation was negative but the Widal reaction positive. It might be supposed that only the agglutinating substance was transmitted to the fetal circulation, but the observation of Jehle in a twin pregnancy (the bacteriologic examination in both being negative, the Widal reaction positive in one) would indi-



cate that a bacillary infection does take place but to such a slight degree that it is not revealed by objective examination. It is very difficult to understand how the resorption of the agglutinating substance could be associated with special conditions which on their part are impregnable to bacilli. This coincides with the generally accepted view of to-day that the Widal reaction is of greater value as regards infection than in immunization. The latest period of a positive Gruber-Widal reaction I recently found in a report of Krusen to be twelve days post partum. Here unfortunately nothing was mentioned as to the condition of the child. If this is to be regarded as a sign of health it would also indicate that the child had recovered completely from the intrauterine infection.

On the part of genuine *croupous pneumonia*, of which Fränkel's diplococcus is regarded as the pathogenic agent, the intrauterine transmission must be looked upon as proven. According to the reports two types of the disease may be differentiated in the fetus. In investigations of still-born infants between the seventh and eighth months (Netter, Birch-Hirschfeld) the disease has been found as a general infection. No particular local change was discovered microscopically at the autopsy. Fränkel's pneumococcus could be demonstrated in the fluids of the placenta by means of smear preparations and by culture. In the fetus characteristic cultures were obtained from the venous blood of the umbilical cord, of the liver, and of the heart. In the animal experiment the cultures were always highly pathogenic. Correspondingly, in infants who were born of mothers ill of pneumonia and afterwards died the pathologico-anatomical lesion was positive. In a newly born child which perished "very soon" after birth Marchand demonstrated a "quite uniform, dense, red hepatization of the right lower lobe and a very profuse pleuritic exudate." Unfortunately no bacteriologic investigation was made. In a case of Birch-Hirschfeld the "entire right upper lobe and the posterior part of the right lower lobe showed dense pneumonic infiltration and a deposit of fibrin upon the pleura" (the infant died in two days post partum). Fränkel's pneumococci, in addition to staphylococci and streptococci, were found microscopically as well as by culture in the pneumonic lesions and in the spleen. The bacteriologic investigation of the organs of the mother, who perished soon after delivery, furnished the same results. Finally, the pathogenicity and identity of these bacteria were demonstrated by the intraperitoneal inoculation of particles of the fetal liver and spleen. Both authors incline to the view that the infection arose during birth.

Of other seldom observed infectious diseases the intrauterine infection of the fetus has been demonstrated bacteriologically in *relapsing fever*, *Asiatic cholera*, *anthrax*, *erysipelas*, *sepsis* and in *malaria*. A pure general infection and the absence of any characteristic local change predominate. Only in erysipelas have the development of endocarditic deposits and purulent peritonitis been observed.

## ASPHYXIA OF THE NEW-BORN

Passing now to the actual act of birth, probably the greatest danger which threatens the infantile life at this time is a *hindrance of the interchange of gases* with the mother. During pregnancy the mother breathes for her child. Within the placenta the oxygen which is absolutely necessary for the life of the fetus, its structure and growth, and the maintenance and activity of the organs, is absorbed from the maternal blood. In the same region the excretion of the products of combustion occurs, especially of carbonic acid which remains from the fetal metabolism. The navel artery conducts the blood that has been utilized through the umbilical cord to the villi of the placenta where it is purified and supplied with new combustive material from the umbilical vein in the infantile body. So long as this passive supply of the child is sufficient for all requirements the fetus remains in a state of apnea, i. e., all substantive respiratory activity is absent. This changes at once when the maternal supply of oxygen shows a decrease or is arrested. The deficiency in oxygen which then occurs in the fetal blood probably irritates the respiratory center on account of the accumulation of readily oxidizable bodies, which otherwise undergo further combustion, and forces the fetus to compensate for the deficiency by the activity of its own respiratory organs. Thus we explain the origin of the first respiration of the child after birth under normal conditions. When the uterus with the greater portion of its contents evacuated, contracts energetically, the placenta is constricted to the smallest extent and occasionally more or less loosened, so that the respiratory space of the fetus is decidedly lessened or completely obliterated. With the first respiration the thorax dilates, air enters the lungs through the nasal and oral openings, and the contained oxygen enters into direct relation with the blood capillaries and their contents. The extensive unfolding of the pulmonary tissue includes an elongation and a dilatation of its vascular net, makes possible a more extensive filling of the same, and hence decidedly increases the introduction of oxygen. With the first inspiration the blood is actually drawn from the right heart into the lungs, the pulmonary artery dilates, the ductus Botalli narrows and contracts. The succeeding expiration forces the blood, which has become arterial, into the left auricle. Therefore, with the first respiration the blood pressure in the right heart falls, is increased in the left heart, and the foramen ovale, on account of its structure and position, closes of itself. From now on the right heart receives pure venous blood, the left blood of arterial character. Although in this manner the lesser circulation receives, with the first respiration, its complete formation and absolute division, the mediate effect upon the fetal placental circulation is not lacking, since through the comparatively great depletion of blood the blood pressure in the umbilical arteries is lowered and they contract. Although the supply of oxygen to the placenta has not ceased entirely it receives a further limitation with the first respiration.



Naturally both occurrences are the more rapid the stronger the respiration after the birth of the child.

The condition is similar when a disturbance of the maternal supply of oxygen occurs prior to death. The fetus makes premature respiratory movements which, on account of the deficiency of air, are without result. It can only aspirate what is directly in front of its nasal and oral openings, therefore fetal fluid, mucus, blood, and meconium. The simultaneous effect upon the inclusion of the lesser circulation and upon the exclusion of the placental circulation, as already mentioned, is rather calculated to increase the already existing deficiency in oxygen. Suffocation must therefore occur if the obstruction is not soon removed and the maternal supply of oxygen reestablished, or the child is not released from its airless prison and placed in a position to inspire air of itself. With non-oxidation of the fetal blood the early irritation is soon followed by a stage of paralysis. In addition to the respiration other centers are stimulated to specific activity through the deficiency in oxygen. The involvement of some of them is of great importance in the clinical estimation of this process since it usually precedes that of the respiratory center, and when it occurs attention must be directed to the condition in time. The best known of these is the passage of meconium. At first the result of a stimulation of intestinal peristalsis, it is facilitated later, in the soporose stage, by the occurrence of a sphincter paralysis. That this symptom is of no value in breech presentation probably requires no remark. In head and transverse presentations it is suspicious as it frequently occurs first and comparatively early. It is true there are cases of contamination of the liquor amnii during birth or pregnancy in which the cause cannot be found subsequently, the child being born alive. More positive criteria are furnished by the condition of the infantile heart sounds. At first, by irritation of the pneumogastric nerve, there is a decrease in their frequency which, provided it continues, is regarded in obstetric practice, as one of the most reliable signals of warning on account of its early appearance. Under normal conditions a transitory decrease during the uterine contractions with strong pains is an ordinary observation, particularly after rupture of the waters. This recuperation in the intervals between the pains indicates that with the relaxation of the muscles of the uterus the danger has passed. Limitation of the respiratory surface of the fetus and the simultaneous encroachment of the maternal blood changes in the intervillous spaces, due to the muscular contraction or great compression of the infant skull, are the causes which are invoked in explanation according to the stage of birth. Continued or even progressively increased slowing of the fetal heart sounds below 100 is regarded in obstetric practice as extremely critical. The infantile life is in the greatest danger. An inspiratory movement has already occurred or is imminent. In the physiologic animal experiment of artificial suffocation this decrease in frequency is first succeeded by a period of transitory increase with paresis of the pneumogastric nerve. In the newly born child and fetus this change, as a rule, escapes observation. The

retardation continues while the contracture of the heart simultaneously loses in power, becoming slower and weaker until it finally ceases. In contrast to this there are a number of observations in which this stage of progressive increase appears alone or is especially distinct and prolonged. Strong compression of the infantile skull and brain in a narrow pelvis is usually given as an explanation. That this symptom must greatly exceed in urgency the decrease in rapidity is obvious from the series of conditions founded upon experiment. Although, with beginning deficiency in oxygen, the action of the heart is altered first, nevertheless, with a lack of oxidation of the fetal blood this organ remains active the longest. If the respiratory center is already affected by paresis we often observe only feeble and infrequent cardiac contractions. But even when these have ceased a renewal, at least at first, is not impossible. The centers are still in a paretic condition which only with a continued deficiency of oxygen, passes into actual paralysis and with this death. Finally it must not be forgotten that infantile muscular convulsions which, even with intrauterine suffocation, precede the paresis of the respiratory center, become obvious to the mother by the succeeding absolute quiet.

In addition to this course of intrauterine suffocation with arrest of the respiratory movements there is also a possibility, according to the interesting statements of Schulze, of a very gradual retention of oxygen in which the differences of degree which are necessary to stimulate the cerebral centers in question are lacking, the child gradually passing into a soporose state which excludes any central reaction. This author has also pointed out that by removal of the obstruction and the reestablishment of the placental circulation the further development of the process can be arrested and the apneic existence of the infantile life, with all of its usual intrauterine functions, can be readjusted to the normal even though respiratory movements have already occurred.

For this symptom-complex originating through deficiency of oxygen the term **asphyxia** or **suspended animation** is generally employed. The former designation is suitable only for the severer forms. Its inclusion of all stages, after the above description, is inconsistent. For all degrees the latter designation is more appropriate and correct.

The question of how or by what means the interchange of gases intrauterine is inhibited belongs to the province of obstetrics. We will, therefore limit ourselves to a simple detail of the factors which come under consideration. First we should consider a disease of the mother which brings with it a deficient oxidation of her own blood—the source of supply to the child: disease of the respiratory organs, eclampsia, disease of the heart, great blood loss, death. In healthy mothers it is usually the consequence of a labor protracted after the escape of the amniotic fluid, due to a constriction of the bony pelvis or an unusual rigidity of the soft parts: anomalous pains, prolonged and rapidly successive contractions of the uterus, *tetanus uteri* or a high position of the contraction ring with the expulsion of the child into the

lower uterine segment. If in these conditions the hindrance of oxygen is more or less indirect and gradual the compression of the umbilical cord acts directly and therefore more rapidly and energetically—either from prolapse or because of its rigid looping—and the premature loosening of the after-birth is observed as in placenta previa, in the birth of the second twin, nephritis of the mother, etc. To this must be added the consequences of a greater compression of the infantile skull and the brain in contracted pelvis, particularly with the rigid and contracted soft parts or with instrumental labor. The pressure upon the brain, by producing irritation of the pneumogastric nerve, causes a slowing of the pulse and with this a decreased circulation of the blood in the fetal placenta. This cause is much rarer alone than in combination with a decrease in the size of the placenta brought about by an immoderate effort of the uterus. If, as is usually the case, this is due to changes which disappear with relief of the pressure, therefore after birth, a permanently increased cerebral pressure may develop in consequence of vascular rupture, either due to a marked overlapping of bones or to fissures of bone and fractures, causing an intracranial effusion of blood. At the convexity of the brain these hemorrhages in the newly born child are as a rule readily sustained; if they are situated at the base, however, there is usually no hope of recuperation on account of the vicinity of vital centers.

According to whether the intrauterine further development of asphyxia has been spontaneously or artificially arrested by labor the infant shows a correspondingly slight or grave degree of disturbance. While an infant under normal conditions is active after birth, soon makes respiratory movements, cries, and presents a bright red color of the skin, the asphyxiated child is motionless, the respiration is absent, slight, or incomplete and associated with distinct râles, and the color of the skin is bluish or a pale gray, cadaverous. In the severe forms a scarcely perceptible palpitation indicates that traces of vitality are present. Many even of the milder forms would gradually succumb if proper measures were not instituted. On the other hand these are most successful the earlier and the more accurately they are employed. Therefore a prompt recognition of asphyxia and of its extent is of great practical importance. If suspicious factors have been apparent during birth or from the nature of the necessary manipulations all doubt is removed by the above-mentioned condition of the child after delivery, especially the absence of regular and thorough respiratory movements. For an exact determination of the stage of the asphyxia the question whether or not there is cardiac pulsation is decisive. The arrest of pulsation, however, may have just occurred, only a short while ago, therefore paresis is present and a quick institution of proper measures may invoke the heart to renewed activity. The special condition of the cardiac action, whether slow, rapid, or irregular, can be utilized just as little as the observation of individual respiratory movements. The prognosis which has been considered favorable on this account has often proved later to be erroneous. Such symptoms are of more value in judging the effect of resuscitative measures. As every advance improves



the prospect, so is the final result characterized by a regular and complete action of both organs. The first and most important point, as we have learned to recognize from B. Schulze's excellent description, is the character of the muscle tonus. The living muscles, even in repose, present a certain resistance and rigidity which prevent their immediate response to every external action, especially gravity. In the cadaver this condition has given place to a general relaxation and lack of resistance. If in the new-born the muscle tonus is retained the prognosis may be regarded as good and the condition looked upon as a mild degree of asphyxia. The color of the skin is then usually bluish red, cyanotic, or livid; its turgescence corresponds to the norm. The heart's action, although occasionally much slowed, is strong. The vessels of the umbilical cord are completely filled and maintain an energetic pulsation. In the beginning there is no respiration but when it occurs it is at first superficial, then deeper, with distinct râles, and associated with a decided involvement of the facial musculature. If the muscle tonus has disappeared the limbs droop and in particular the lower jaw falls; if upon raising the child the head sinks flaccidly to the side or backward we are dealing with a severe form, a deep stage of asphyxia. The prognosis is doubtful, rather bad and unpromising. The skin is generally grayish, wrinkled, cadaverous. The action of the heart, which may be either rapid or slowed, is weak and very indistinct. The umbilical cord is pale, empty of blood, collapsed, and often stained a light, yellowish green by meconium. Pulsation in the umbilical cord is either absent or very slight. In this stage the respiratory movements are observed a few times, but they are superficial, without involvement of the facial muscles and with marked retraction of the epigastrium and of the lateral portions of the thorax. Of great prognostic importance is the reaction of the muscles of the palate. In the active child this contracts at once upon introduction of the finger, but with deep somnolence this effect does not occur; there is a flaccid yielding to the pressure of the finger, the mouth and the pharyngeal cavity gape and the finger may penetrate deeply into the pharynx without resistance.

The prophylaxis of asphyxia acquired intrauterine belongs to the realm of obstetrics. A precise knowledge of the normal, the early diagnosis of any abnormalities and the complications to be expected therefrom, that is to say, their skillful relief—briefly, the teachings of the special pathology of midwifery and its therapeutics—are the points which give us the necessary aid. In all of the measures which have come under consideration the obstetrician must always reflect upon the dangers to which the maternal life is subjected. Only this consideration, associated with the fact that the danger of suffocation of the child will be increased by the operation, and particularly the more difficult this be, insures a proper decision and prevents those unfortunate failures which also for the mother may have an uncertain or even negative result.

**Treatment.**—A methodical treatment of asphyxia of the new-born dates from the middle of the last century. The knowledge of its origin and develop-



ment, instituted and enhanced by the investigations of Cazeaux, Krahmer, Hecker, G. Veit, Hoogeweg, Schwartz, d'Outrepoint, Volkmann, Bärensprung and especially of B. Schulze, has furnished the scientific basis by which the previous planless and often improper method has been directed into clear channels. The principal factors of suspended animation set forth by the last author and the indications arising therefrom have been maintained to this time. Their knowledge is a necessary prerequisite for a successful therapy. The most important sequel of deficient oxygen is the increasing soporose condition of the medullary centers which causes the respiration after birth to cease and the circulation to become less and less forcible. In addition to this is the occlusion of the respiratory passages by aspired masses when respiratory movements have occurred intrauterine. With the idea that the free entrance of air into the lungs must be made possible some authors (Schroeder, Ols-hausen, Veit, Ahlfeld, etc.) regard the removal of the last-named deleterious effect as the first requirement. This is accomplished, as is well known, by suction by means of an elastic Nélaton catheter. B. Schulze believes this to be superfluous. By the swinging movement advised by and named after him, which simultaneously fulfills the other requirement to increase the circulation and respiration, the removal of inspired material occurs "naturally and much more completely." During the expiratory phase the bronchial tubes and their branches are compressed, and the substances thus forced into the upper respiratory passages and readily expectorated. By suction only the upper respiratory passages can be emptied. There the inspired material produces râles but does not hinder respiration. The latter occurs in the bronchi and their branches which are not influenced by the suction. He also regards the prolongation of this measure as an unnecessary loss of time, which may readily be serious on account of the steadily increasing sopor of the central organs. Admitting all of Schulze's arguments to be correct, a brief suction of the upper respiratory passages nevertheless appears to me to be proper. By Schulze's method alone, without previous catheterization of the trachea, the contents of the nose and oral cavity are expelled. The expulsion, however, is greatly facilitated and occurs more rapidly provided the upper respiratory passages have been cleared. Also there are occasionally tenacious mucous substances which cannot be blown out of the catheter without much difficulty, therefore their removal by simple compression of the bronchi and the aid of gravity is scarcely possible. In short, practical experience has taught me that a brief aspiration of the inspired material is decidedly advantageous. For this purpose, however, the emptying of the nasal and pharyngeal cavities is no less important—a point which is but little or not at all mentioned in text-books. This is best and most rapidly accomplished by aspiration with a catheter. In man, and particularly in the new-born infant, the respiration is predominantly nasal. That with restricted space a displacement of the physiologic process by the aspired substances may actually take place is readily conceivable and in the case in question is very evident from the amount of mucus which is discharged. Here an absolute hindrance of

respiration certainly never occurs. The mouth is a sufficiently wide expedient. Nevertheless in the course of years I have not been able to escape the impression that the observation of this precautionary measure is of value, particularly in promoting a regular, powerful respiration. These preparatory manipulations of course must not take too long nor be continued unnecessarily. A single aspiration through each of the nasal openings from back to front and one or, at most, two suction of the trachea are usually sufficient. The time required for this, provided everything has been prepared before the labor, is but a few minutes, so that if anything at all is to be done for the asphyxia the time can scarcely be regarded as a serious obstacle.<sup>1</sup>

However, the arguments set forth by B. Schulze are neither absolutely without objection or entirely conclusive. First of all, the swinging advised by him necessarily begins with a dilatation of the thoracic cavity—a point to which Schroeder, Olshausen, Ahlfeld, and others have called attention. The child is grasped by the shoulders, held downward, and then swung so far upward that it doubles upon itself, with the feet and back toward the physician. The expiratory compression of the thorax, therefore, begins at the earliest with the bending of the body upward against the diaphragm. Until then the distention of the thorax prevails and therewith also of the lungs, hence a deeper aspiration of the previously inspired mucous masses is possible. It follows, therefore, that the removal of these foreign substances is necessary, in so far as possible, prior to the beginning of Schulze's swings.

Naturally, the essential point of the entire treatment is to increase respiration and circulation. The regular ventilation of the lungs, i. e., the methodic introduction of air which contains oxygen and the removal of exhausted air, is the best measure for successfully counteracting the diminution of oxygen in the blood. On the other hand also, in so far as the physiologic process is fulfilled by an inspiratory dilatation and an expiratory contraction of the thoracic cavity, this acts as a stimulant to the circulation of the blood and therefore furthers the introduction of oxidized blood into the central organs and aids essentially in awakening them from their soporose condition and stimulating them to normal activity. In the slighter grades of asphyxia with retained muscle tonus it is usually possible to abbreviate this course of development as the central organs still react to strong irritants by spontaneous respiratory movements. The above-mentioned aspiration of the upper air passages with a catheter has an excellent effect in this direction—another reason why the procedure should not be omitted. Other measures that have been successful are friction of the back, hot baths, cold affusions, especially to the nape of the neck and the epigastric region. In the severer grades of asphyxia the proof of an absence of muscle tonus indicates that external irritants are ineffectual and useless. Slapping, friction, or whatever else of this sort is attempted with the child is an unnecessary loss of time. Here

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<sup>1</sup>“But a few minutes” is an eternity in the treatment of asphyxia. Every second passed without respiration is a danger to life, or what is worse, a cause of mental inferiority, epilepsy, or paralysis.—EDITOR.



artificial respiration alone—the above-mentioned regular ventilation of the lungs—is of help. Naturally this method must also be employed in the lesser grades if other measures prove ineffectual. Among the methods which come under consideration two are at present of equal prominence: insufflation of air into the lungs, and Schulze's swinging movements. The former, especially recommended by Hüter, Schroeder, and Olshausen, can only be attempted with the catheter introduced into the trachea. Direct insufflation of air from mouth to mouth or from mouth into the nose leads to inflation of the stomach and the contiguous intestines. The hindrance thereby in the movement of the diaphragm, that is, the forcing upward of this structure toward the thoracic cavity and the slowing of the heart associated with it, have rather an unfavorable effect upon an eventual resuscitation. Insufflation of air through the catheter is principally a substitute for inspiration, expiration is induced by a manual compression of the lower aperture of the chest which simultaneously aids the upward movement of the diaphragm and therefore must be directed backward from above. The chief danger of the insufflation of air lies in the laceration of the pulmonary tissue and the development of an interstitial emphysema, which, by its special distribution in the hilus and thence into the anterior mediastinal space, obstructs the distention of the lungs. The strength which is permissible in inflation is very difficult to determine. During the period of my assistantship I can remember several marked examples of this accident at the autopsy. The operators were physicians. In the attempt to demonstrate the method upon the cadaver the same accident has occasionally happened to me, in so far at least as the beginning of this change was detected later. But aside from this complication, according to B. Schulze, the insufflation of air is to be limited alone or principally to the respiration. The intrathoracic pressure upon inspiration is increased as is the succeeding expiration. By this deviation from the normal the simultaneous effect upon the circulation is decidedly limited if not altogether illusory. Practical observations as well as theoretic considerations have inclined me seldom to employ insufflation of air as an aid to artificial respiration. Even in those cases in which B. Schulze advises it—in premature infants with very soft thoracic walls—I have not convinced myself of its value. Here the insufflation of air is of all the less worth as the insufficiency of the chest wall is not relieved but, on the contrary, immediately reappears, and the possibility of an efficient spontaneous ventilation again comes into question.

Very important and worthy of consideration for application in general practice is the question which Reich has raised of an ultimate transmission of tuberculosis by this method. Within thirteen months ten children perished from tuberculous meningitis where a tuberculous midwife had given aid during labor. She practised the resuscitation method by the inflation of air from mouth to mouth. The use of the catheter would obviate this danger but little. Accordingly the suction of mucus from the upper air passages must also be highly improper. In addition to tuberculosis the various forms of infectious angina, influenza, etc., are no less likely. The danger of infec-

tion is not entirely eliminated if insufflation and the suction of mucus are omitted. The intimate contact of the physician or midwife with the mother and the child offers sufficient opportunity for transmission. Such persons should be prohibited from practising. This assertion is upheld by the recent views of Martin and Rosthorn, according to whom follicular angina of the physician or of the nurse may very readily give rise to puerperal disease in the lying-in woman. The unimportance of finding a method which will permit these persons to give assistance notwithstanding their disease, but rather their complete abstinence, in which lies the greatest safety for the patient, is also applicable to the method of insufflation of air. Nevertheless, if I am opposed to this method it is only because it appears to me to be inadequate and that better methods of artificial respiration are at hand.

In the mature infant, or one born near term, with a thoracic wall not too pliable, *Schulze's method* has proved to be essentially better and more reliable. This most closely approaches the normal respiratory movements. Although the general principle of Schulze's swinging movements is well known, it may nevertheless be advisable to enter somewhat in detail into the essential factors of the same since deviations are not infrequently encountered in practice which show that a complete understanding of the importance of this method is lacking. On account of the simplicity of Schulze's description the following is quoted literally from his book "Suspended Animation of the New-Born."

"After the umbilical cord has been severed the infant is grasped at the shoulders, between the two hands, so that the thumbs rest against the anterior surface of the thorax, the index fingers in the axillæ, and the remaining fingers across the back. The head, which would otherwise fall flaccidly, finds a comfortable support between the ulnar borders of the opposed palms of the hands. In this manner the obstetrician, who stands with somewhat separated legs and slightly inclined upper body, holds the child in front of him with his arms extended downward. Without a pause he swings the extended child from this hanging position upward. When the arms of the obstetrician are raised somewhat above the horizontal he halts the movement so gently that the child's body is not thrown forward but slowly sinks forward and the weight of its pelvis strongly compresses the abdomen. The entire weight of the child at this moment rests upon the thumbs of the obstetrician which are upon the thorax. Special care must be observed not to compress the thorax by the grasp with which the child is held; the body rests with the floors of the axillary cavities upon the index fingers exclusively. There must not be either a lateral compression of the thorax, although the ulnar borders of the hand offer a firm support to the head, nor must the thumbs press the thorax anteriorly. In the upward swing a flexion of the vertebral column in the thoracic area should not occur. This must take place almost exclusively in the lumbar region; the thumbs, even now, should not make decided pressure upon the thorax but should only form a support for the infant's body, which slowly inclines forward. The uplifting to the horizontal is accomplished by a powerful swing of the arms at the shoulder-joints, then the rais-



ing of the arms must gradually become slower and by carefully measured movements in the elbow-joints and movements of the shoulder-blades upon the body the operator regulates the gradual inclination of the lower part of the child's trunk. By this gradual forward inclination of the child's pelvis over the abdomen considerable compression of the organs of the thorax, on the part of the diaphragm as well as of the walls of the chest, takes place. After the forward inclination of the child has been completed the obstetrician moves his arms downward between his extended legs. The body of the child is thus extended in one swing; the thorax, free of all pressure (the thumbs of the obstetrician now lie quite loosely upon the anterior wall of the chest), will now dilate on account of its elasticity, but more particularly because the child rests with its upper extremities upon the index fingers of the obstetrician and the sternal ends of the ribs are fixed thereby, the weight of the body with a quite decided swing being utilized in raising the ribs. The diaphragm also inclines downward with this swing, as is noted by the contents of the abdominal cavity. Thus a decided inspiration results in a purely passive manner. After a pause of a few seconds the child is again swung upward to the previous position, and, when it slowly inclines backward with its entire weight upon the thumbs which press upon the anterior wall of the thorax, mechanical expiration results anew."

It is of great importance that the child is loosely encircled so that the thorax is not compressed and constricted in the changes which take place. This is particularly necessary in the inspiratory phase in which all results would otherwise be impossible. Sometimes the head falls forward unnoticed and by bending limits the introduction of air. Finally, throughout B. Schulze's description there is the particular caution that all swinging should be "gentle," not forcible, but skillful. During this swinging great external force may accidentally be exerted upon the organs of the abdomen, the walls and the contents of the thoracic cavity. Aside from fractures of the ribs and of the clavicle, in the autopsy reports injuries to the internal organs are mentioned to which the death of the child was referred. Runge observed effusions of blood in the adrenals from the size of a cherry-stone to that of a hazelnut, also a complete destruction of the upper lobe of the left lung by an enormous hemorrhage with rupture of the pleural covering. Winter reports a case of rupture of the liver with hemorrhage into the abdominal cavity and another of complete separation of a syphilitically enlarged spleen. Körber and Koffer report similar observations of subserous hepatic hematmata and rupture of the liver with effusion of blood into the abdominal cavity. Gebhard reports the tearing of the pleura over the head of the second rib with hemorrhage into the thoracic cavity. B. Schulze strongly objected to the view that these unfortunate accidents were the result of his method. Subserous and parenchymatous effusions of the liver, to which Rokitansky, Förster, and Weber called attention prior to Schulze, are common and quite typical findings of severe and fatal asphyxia, and their development is the direct consequence of the constantly increasing congestion in this organ. Schulze's method acts in oppo-

sition to this, for in addition to stimulating the activity of the lungs it mechanically aids the circulation of the blood, particularly that which accumulates in the veins, corresponding to the position of the cardiac valves, by forcing it into the arterial circuit, thus making possible a further compensation in the capillary tracts in the lungs as well as in the rest of the body. It is true, a tense hematoma may rupture as the result of shock to the body. That it may, however, resist the swinging is illustrated by a case of Koffer's. Here rupture took place later, upon the seventh day, and most likely, according to B. Schulze, in consequence of an injury at the base of the skull; respiration again became difficult and caused a renewed hepatic congestion. These and similar hematomata of other abdominal organs may be regarded as unfortunate and unpreventable complications which have no causal relation with the swinging.

A second series of unfortunate cases is referred by the authors themselves to their forceful management of the child. Chief among them are fractures of the ribs and of the clavicle; but injury to the organs is also possible. In addition to the marked swinging over the abdominal side in the expiratory phase, particularly with heavy children, the absolutely necessary upward swinging appears no less dangerous when the body falls back again, particularly if the obstetrician does not at once note this error and instead of permitting the body to decline holds it up, although only for a brief time, by the shoulders. As is shown by the consequences of corresponding trauma in adults, the parenchymatous organs, rich in blood (liver, kidneys), which are situated in the posterior part of the abdominal cavity, are hereby especially endangered. Such injuries, however, can be avoided by a cautious employment of the method. If to this we add that these accidents, in comparison to the frequency with which Schulze's method is practised, are extremely rare, there can be no objection to the employment of the method in a given case, naturally, with the necessary prerequisite that the practical application and the theoretic comprehension have been successfully worked out.

Another method of artificial respiration is *faradization of the phrenic nerve*, first mentioned by Bär and Pernice. This is limited to the periodic production of inspiratory contractions of the diaphragm. To prevent a still deeper suction of the aspirated material the previous use of the catheter in the air passages is necessary. In the severer grades of asphyxia of the newborn a sufficient dilation of the thoracic cavity by contractions of the diaphragm is very much questioned by B. Schulze on account of the complete relaxation of the remaining muscles of the body and the ready flexibility of the ends of the ribs. Besides, there is no stimulation to expiration, the most important motive power of intrathoracic circulation of the blood. Finally, the obstetrician would seldom have the necessary apparatus in working order, nor is it possible that he would always have it at hand.<sup>1</sup>

<sup>1</sup> The editor cannot but deem the last sentence a little naïve. The following from A. Jacobi, *Therapeutics of Infancy and Childhood*, 3. ed., page 84, may be found welcome. Electricity was recommended in cases of asphyxia as early as 1793 by Hufeland. But the



The best and most successful methods for resuscitation in use in adults, Marshall Hall and Sylvester's methods, are of much less value in the new-born than Schulze's swings. Marshall Hall places the infant upon its abdomen and first turns it slowly to the lateral position or a little beyond and then rapidly back again upon the abdomen. These movements are repeated about fifteen times per minute. Compression of the thorax by means of the body weight enforces expiration in the one phase while, with an arrest of the pressure, inspiratory dilation of the thorax occurs in consequence of the elasticity of the walls. The first action is opportunely aided by pressure upon the back between the shoulder blades or on both sides of the thoracic wall. Sylvester forces the inspiratory distention of the thorax by decided tension of the muscles of the arm and thorax, particularly of the pectoralis major. The infant is laid upon its back and somewhat elevated at the shoulders. While an assistant holds the feet the obstetrician slowly abducts the arms arch-wise to the head. The expiratory emptying of the thoracic cavity occurs by rapid adduction of the flexed arms to the sides of the thorax which simultaneously is slightly compressed. These manipulations also are repeated about fifteen times per minute. To prevent the tongue from falling back and closing the

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first case in which the rhythmical faradization of the phrenic nerve and its associates was resorted to (by Ziemssen) for the purpose of producing artificial respiration was that of an asphyctic girl poisoned by carbon oxid. The phrenic nerve acts on the diaphragm. Its aids are the cervical plexus, which controls the trapezius, levator scapulæ, and middle scalenus muscles, and the brachial plexus. The ramifications of the latter are the anterior thoracic nerve for the pectoralis major and minor; the posterior thoracic for the middle scalenus posterior superior serratus, and the rhomboid muscles; and the lateral thoracic for the serratus anticus major.

In asphyxia of the new-born, the systematic faradization of the phrenic nerve was first employed by Lauth and Pernice.

The point of application selected by most authors is near the sterno-cleido-mastoid muscle, over the phrenic nerve. The other pole is applied either to the neck or to the diaphragmatic region or any other part of the surface. The localization of the effect to the phrenic nerve alone, which was insisted upon by many, is certainly an illusion. The current will surely strike the pneumogastric, phrenic, sympathetic, and many sensitive and motory nerves at the same time. As this cannot be avoided, as indeed it is better that it should be exactly so, it is best to use large sponge electrodes and moisten them thoroughly with salt water. The head, arms, and shoulders should be slightly raised, and a small pillow placed between the shoulders for the asphyctic baby to rest on. One of the electrodes must be kept stationary, the other brought into contact with the surface *a single moment only*. A deep inspiration will then take place, the lungs will expand, and lateral pressure on the lower part of the chest should be resorted to for the purpose of emptying the lungs afterwards. Another application is then made with the same result, and must be followed by the same manipulation. This has to be continued until the baby cries, and until it appears safe to discontinue the application. Whenever a cough or a coughing movement is noticed, it should be omitted temporarily. The favorable result is, however, not always permanent. The causes of the asphyctic condition are still active, and the infant will require resuscitation again, and perhaps many times. That is why close attention must be paid, sometimes for hours.

Great care should be taken in regard to the duration of the application. Continued or too frequent irritation by the current *causes over-irritation and paralysis*. Not infrequently the immediate effect is a favorable one, inspiration becoming deep and the

larynx that organ should be drawn forward. Behm, Champineys, and Schauta have tested the effect of these methods on the cadavers of infants and compared them with Schulze's swings. The first two authors reached the conclusion that Sylvester's method is decidedly preferable. Schauta, on the other hand, obtained five or six times better results with Schulze's method and accounts for this deviation by an improper performance of the swings by the other two authors. Sylvester's method has the great disadvantage over Schulze's swings of absolutely demanding an assistant and is therefore decidedly less suitable in practice. This is also true of the less effective Prochownich method, in which the child is held upright by the legs by a second person while the obstetrician compresses the thorax.

Lah's method—periodic flexion and extension of the trunk of the child, who is held by the buttocks and shoulders—may be well employed in the warm bath to aid respiration. Schroeder's proposition appears to be even better: After flexion over the anterior side, to somewhat hyperextend the infantile trunk by slightly raising the back of the child with the supporting hand.

*Rhythmical contractions of the tongue*, practised manually with a cloth or forceps and first advised by Laborde, were once much employed. Stimula-

heart active, but after a short time the former grows more superficial, the pulse feeble, and the cyanotic hue returns to the lips and finger-nails. Then it is time to stop for a while, and resort temporarily to other means of resuscitation. The practice of Lauth, who applied the current persistently for two or three minutes, is decidedly improper and dangerous.

In some cases, where the interrupted current is ineffective, the galvanic (continuous) current, with occasional reversions, has been known to yield better results. In my own cases I have never had an opportunity or been under the necessity of employing it.

The application of large sponge electrodes may not always be convenient. In those cases no harm is done by using the metal poles instead. Though the irritability of the brain (and nerves) is low in the newborn, the pain produced by the interrupted current thus applied is very intense, and the effect on the contraction of the diaphragm quite marked. That is why it is not necessary to lose time in preparing, if it be not on hand, the more complicated apparatus. Still, exhaustion is more readily obtained through resuscitating by pain and muscular action combined than by muscular action alone. In most cases, however, I was satisfied with not losing even a fraction of a minute, particularly in those early times, when the most convenient apparatus was the old-fashioned rotating machine.

How long is the asphyctic baby to be watched and the attempts at resuscitation to be renewed? At all events, they must not be given up so long as the heart-beats are audible, though ever so feebly. Nor is a single scream sufficient to permit watchfulness to be relaxed. The deep recession, during inspiration, of the diaphragmatic region (the peripulmonary groove of Trousseau) should have ceased, the cry be vigorous, the eyes wide awake, and the extremities in lively motion. Before this is accomplished there is danger of a relapse, partly from impaired innervation and the continuation of some of the causes of asphyxia, and partly from obstruction by mucus, which may be coming up constantly and gather in the pharynx and posterior nares.

Some cases of asphyxia are particularly troublesome; those in which it is due to prematurity of the newborn or to an actual anatomical change (hemorrhage, compression of the brain or medulla) which requires time to get well or will terminate fatally; also those which are due to congenital anomalies of the organs of circulation or respiration (syphilis of the lungs, effusion into the pleural or peritoneal cavity, thoracic tumors, etc.).—EDITOR.



tion of the nerves of the palate, pharynx and tongue was supposed to produce reflex respiratory movements. As already mentioned, the reflex of the pharyngeal and palatine muscles is the last to disappear. Therefore success is to be expected only so long as this reflex is retained, i. e., in the first stage of asphyxia. Later, all such attempts would cause an unnecessary loss of time.

In conclusion, in a brief review of the methods for the treatment of asphyxiated new-born with a special consideration of their differences in grade the following arrangement will prove serviceable. The first requirement is the proper preparation of all the necessary utensils for resuscitation. A plentiful supply of hot water and the warming of all diapers should be the first care and the strict duty of the nurse at every labor. It will be well, however, for the obstetrician to convince himself that everything is in readiness so that at any moment (particularly before an operation) the required articles will be at hand. After delivery every moment is valuable and any oversight may cause a fatal loss of time in the case of an asphyxiated child. A lack of this precaution makes the physician just as liable as the nurse, perhaps even more so. Of equal importance is the previous preparation of a place to lay the child after the cord has been severed so that it may be handled. A simple table supplied with a hard pillow or with a blanket folded several times and covered with fresh linen or a towel, is usually sufficient. Upon this one or two catheters and a few warm diapers or towels are to be placed. In addition a hot bath at a temperature of about  $40^{\circ}$  C. should be prepared and alongside of this a vessel containing cold water. For a quick control the bath thermometer had best be placed in the bath-water. Naturally, there should be an additional plentiful supply of hot water so that with a prolonged operation the bath may be kept warm. If the child does not breathe after birth the pulsation of the heart must be immediately noted. If this is normal and strong, not slowed, and if the child reacts to blowing by contraction of the facial muscles, the employment of slight cutaneous irritants (friction of the back, quick slaps upon the buttocks, sprinkling with cold water) will soon bring about respiration and cause the child to cry. There is then no necessity for haste in tying the umbilical cord; it is better to wait until the pulsation in the cord ceases. If the child does not react to blowing in the face, if the pulsations are slow and weak, the cord should be tied at once. The degree of asphyxia is quickly determined by introduction of the finger to the root of the tongue. If a contraction of the palatine and pharyngeal muscles occurs the stage is mild and the asphyxia can be overcome. The catheter is introduced into the nasal opening and the pharynx cleared, then the cavity of the mouth, and if respiration has not yet occurred, also the trachea. If the respiratory movement still fails to appear the child should be placed for a short time in a hot bath of a temperature of  $36^{\circ}$  to  $38^{\circ}$  C., the action of the heart observed, and the thorax rhythmically compressed a few times according to Schroeder's method. If all of these measures are without result Schulze's method must be practised at once. After this has been repeated from eight to ten times the

child should be placed for a short time in a hot bath to prevent too great cooling, the respiration and circulation are observed, and the former reinforced during the bath by the method proposed by Schroeder. If the cardiac activity has been increased, if it has become more rapid and stronger, if the skin begins to redden, or superficial respiration, even though quite irregular, is noted, the time has arrived for the successful employment of the previously mentioned cutaneous irritants (friction, cold ablutions). If inspiration occurs during Schulze's swings their performance is discontinued and the child is at once placed in the warm bath for further observation. Naturally all of these measures must be continued until the respiration is sufficiently deep and regular and the child begins to cry, or until there is evidence that further effort will be useless, therefore that the activity of the heart is decidedly lessened or altogether lost. This is usually apparent within one or two hours at the latest. The permanent although slow increase of cardiac action is the only favorable sign; the improvement is often transitory. Unfavorable indications are the complete arrest of spontaneous respiratory movement or occasional spasmodic inspirations with marked retraction of the lower portion of the thorax. These are purely muscular spasms which have no connection with the therapeutic measures and have been proven to have no influence upon the amount of air contained in the lungs. Correspondingly their appearance coincides with a decrease in cardiac activity. Thereafter further attempts at resuscitation are quite hopeless.

This intra partum asphyxia is contrasted by some authors with the second *form occurring after birth*. Placental respiration is entirely undisturbed and not until complete development does the impossibility of a substantive activity of the respiratory organs arise. The cause may depend primarily upon developmental anomalies, therefore, as already set forth, it may be due to defective development of the central nervous system, of the heart, of the diaphragm, of the walls of the thorax, or to the incomplete structure of premature infants. There are reports of compression of the trachea by congenital goiter or a particularly marked hypertrophy of the thymus gland, but in many respects the latter effect is still very doubtful. Local diseases mostly of syphilitic nature and developing intrauterine must also be considered: pneumonia alba, bilateral pleuritic effusions. An enormously enlarged liver may decidedly limit the movements of the diaphragm. Finally, the results of injury to the skull during birth must be mentioned (narrow pelvis, forceps, extraction of the head), the respiratory center being directly or indirectly involved by a simultaneous effusion of blood.

The clinical symptoms of post partum asphyxia are very similar to the form acquired intrauterine, namely, feeble or absent respiration with a still palpable cardiac contraction. Usually the process is such that after a few attempts, sometimes superficial, sometimes very spasmodic, the respiration ceases, or its irregular type is maintained for a time with long interruptions. On account of the undisturbed intrauterine course the respiration is free of râles, but this may also be the consequence of ordinary asphyxia.

Cardiac action, at first normal and strong, soon takes part in this decline, becoming slower and weaker, perhaps with a transitory increase. The other early symptoms usually correspond to the characteristic first degree of intrauterine asphyxia and pass rapidly or very gradually, according to the cause, into the second or paralytic stage. The most important point in diagnosis is that close observation of the course of the labor reveals not the slightest cause for the development of this disturbance. Apart from the similarity of the clinical course the fundamental causes, as mentioned above, are usually unrecognizable. The only exceptions are in the case of developmental defects with distinct external deformity, and especially in premature infants. Accordingly none of the other cases are regarded as asphyxia in the ordinary sense, the etiology being cleared up post mortem. The prognosis depends upon the ability of removal of the underlying cause, and therefore in the great majority of cases may be regarded as unfavorable at the onset. Among the conditions from which recovery may be anticipated, in addition to the slight grades of prematurity, we must include congenital goiter and hypertrophy of the thymus gland, provided the general condition of the child permits of operation. The prognosis may be determined by the degree of the disturbance and the rapidity of its increase. In treatment all of those previously mentioned factors come under consideration which are of service in stimulating respiration and circulation. With a rapid cessation of respiration the severest treatment, Schulze's swings, must be employed; in the milder grades we may limit ourselves to the warm bath, cold affusions, and friction of the skin. That external deformity cannot be treated in this manner need scarcely be stated. The special treatment of premature infants and the eventual prophylaxis have already been explicitly described. In congenital goiter and hypertrophy of the thymus gland only operative removal (extirpation or resection with eventual fixation of the pedicle to the sternum) can be considered for a permanent cure, of which a few excellent examples have been submitted, although from a later period of life (König, Rehn).<sup>1</sup>

## TRAUMA DURING BIRTH

We now come to a description of **trauma**, to which the child is exposed **during birth**. The greatest number of these accidents are due to narrow pelvis. With a spontaneous labor the injuries are usually of a mild nature and are rarely dangerous to life or to health. The severest trauma results from artificial delivery by means of forceps, version, or extraction. The most common lesions are of the head. Its preponderance in size is not rarely

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<sup>1</sup>From much experience with enlarged thymus glands occurring during (lymphatism) later months I suggest careful attention to the posture of such babies. The thymus will fall backwards when the baby is on its back, and relieve the trachea when in a less recumbent posture. For many years past I could demonstrate the presence or absence of an extensive dulness behind and below the manubrium sterni in their connection with the posture of the child.—EDITOR.



increased by an inelasticity. Even with simple labor pains the pressure of the descending head against the promontory of a flat pelvis, and rarely also against the symphysis, may give rise to contusions of the skin which are designated obstetrically as *pressure marks* or pressure streaks. Of oval or more circular shape the former have their typical localization upon the posterior portions of the parietal bone corresponding to the angle of the great fontanelle. From here the pressure streaks pass to the temporal region, usually parallel with the coronal suture, more rarely in a concave direction anteriorly. If during the descent of the head the face is also exposed to strong pressure a pressure streak may be found with slight deviation to the region of the ear and even to the cheek. A single pressure mark is always due to the promontory. If it is found upon both sides of the head the slighter one is due to pressure against the symphysis or to a neighboring area. It is then found either upon the frontal bone or in the region of the eyes and cheeks. From a comparison of spontaneous labor with that terminating artificially we know that the consequences of this compression of tissue are influenced less by the force than by the duration of the pressure. All kinds of injury may be observed, from simple redness of the skin with slight desquamation of epithelium to more or less intense suggillations and necrosis. The time of recovery accordingly is subject to great variation. All traces of the milder injuries have disappeared after two or three days, but for the complete healing of the more extensive lesions a long time is required, with demarcation, formation of granulation, and cicatrization. If the injury extends to the periosteum and becomes necrosed an adherent cicatrix remains which is distinctly visible in later life and occasionally very sensitive to pressure. None of these lesions, even of more severe grade, as a rule leaves any deleterious effect upon the child if the necessary cleanliness is observed and infection prevented. In the main, therefore, the treatment is limited to the employment of indifferent or mild disinfecting salves (boracic acid vaselin, zinc vaselin, and dermatol vaselin).

From these typically situated pressure marks the cutaneous changes must be differentiated which are caused by the tearing of *amniotic* or so-called *Simonart's bands*. Of round or occasionally more irregular form, they at once present defects which, free of necrosis or demarcation, are limited to the superficial layer. If the separation occurred shortly before birth the wound appears quite fresh, as if made with a chisel. Otherwise it may already be granulated or even in a cicatrized condition. Its localization is not subject to special conditions, but may occur anywhere upon the infantile body. In the numerous examples at hand special preference is perhaps shown by the region of the occipital protuberance. In the treatment of wounds which fail to heal the ordinary surgical measures must be employed.

Finally, direct contusions of the skin may result from *mechanical extraction* in those areas where the apices of the forceps have been applied: upon both temples when the head is grasped transversely; when grasped obliquely, on one side upon the forehead above the eye, on the other behind the ear.

Usually the damage amounts to a simple redness with desquamation of the epithelium and disappears upon the second or third day. Hemorrhagic sugillations and deep necrosis are rare and only the result of too great compression by the forceps. With an oblique application of the forceps to the head one part of the instrument may be placed directly over the eye and a moderate pressure may cause an edematous swelling of the upper eyelid. With strong pressure there may be compression and laceration of the organ. Pressure of the forceps behind the ear very often damages the facial nerve at its point of exit from the stylo-mastoid foramen (*facial paralysis*). A very disagreeable situation arises when the ear is enclosed by the forceps and fixed in such a manner that removal of the instrument prior to birth of the head becomes difficult or impossible. Under such circumstances unskilled and forcible attempts at removal may lead to very serious injuries of the external ear and its insertion. The measures to prevent injury to such soft parts by the forceps are provided by the rules for their proper use. The treatment of facial paresis will be considered later.

A special position among the injuries to the soft parts of the infantile head is assumed by the typical accumulation of blood between the pericranium and bone, the so-called *cephalhematoma* κατ' ἐξοχήν. Although with narrow pelvis this results occasionally from a traumatic lesion of the bone, especially a fissure, in the majority of cases it occurs in spontaneous and even easy and rapid labor. Some authors regard its development, similar to that of the ordinary tumor of the head, as the result of pressure during birth, causing a hyperemia of the infantile skull which is increased by a simultaneous asphyxia or complicated by a greater tendency to tearing of the vessels. They base their opinion principally upon the fact that every head presentation shows subperiosteal hemorrhages of mild degree in the course of the deepest segment. This, however, leaves completely unexplained the development of cephalhematoma in the head in a breech presentation. The explanation of Fritsch is more comprehensive and more plausible. According to this author a repeated displacement of the soft parts upon the bone within the loose subperiosteal tissue layer, complete enough to cause not only torsion of the vessels but also their rupture, is responsible. Such displacements result when the head of the child lies firmly against the wall of the maternal birth canal but is nevertheless movable to the extent that it is forced forward during the pain and in the succeeding pause again recedes. This latter factor is of special importance as the scalp may be secured in its original position by adhesion at some point of the maternal soft parts. According to Lange this change is particularly favored by the second and third phases of the mechanism of birth, the anterior rotation of the occiput. In the same manner, with the descent of the head a displacement of the bones may occur by a firmer adherence of certain portions of the scalp than of others to the maternal soft parts, whereby a cephalhematoma originates. By this explanation statistical findings, according to which cephalhematoma

is most common upon the anterior parietal bone in head presentation and upon the occipital bone in breech presentation, are readily comprehensible. Its rarity with a narrow pelvis is explained, on the one hand, by the limitation or hindrance of these periodic to-and-fro movements, due to the disproportion between head and pelvis; on the other hand when a loosening of the scalp does exceptionally occur there is a possibility that owing to the continued compression of the infant's head the injured vessels become thrombotic even before the birth is terminated, and thus an effusion of blood is prevented. A second cause of development, no less important than the mechanical, is also probably the result of circulatory disturbances such as are produced by an asphyxia *sub partu*. The venous vessels of the head and therefore of the bones are completely filled and tend more readily to rupture. Although the exit of blood is primarily aided in this manner nevertheless by a surcharge of carbonic acid its chemism is altered, in consequence of which it remains fluid, as is usually the case in cephalhematoma, instead of coagulating (Spiegelberg, Ahlfeld).

The cephalhematoma occurs as a tumor the size of a nut, apple, or fist, distinctly fluctuating, at first tense, later somewhat undulatory, painless, never passing beyond the limits of a suture or a fontanelle. This last peculiarity is due to its subperiosteal location, the periosteum being firmly adherent to the lateral margins of the bones. Cephalhematoma occurs in not quite 0.5 per cent. of the births. The bone most frequently affected is the parietal, and next in order of frequency are the occipital, frontal, and temporal bones. The condition is usually limited to one bone, but two, three, or, as was observed by Schroeder, even four may be involved. If both parietal bones are involved the suture lying between is retained for the reason previously mentioned. Occasionally, directly after birth the cephalhematoma is covered by a simple edematous swelling affecting the pericranium and does not become noticeable until the second or third day, after absorption has taken place. In cases of this kind the cutaneous covering is at first immotile and cannot be raised in folds until later. In other instances the effusion of blood, notwithstanding the necessary preparations for it have occurred intra partum, may not appear until after birth. The tumor is usually at first of moderate size but subsequently enlarges. This may continue to the end of the first week. After a brief stationary period a gradual decrease occurs which occupies on an average from ten to fifteen weeks. This is accompanied, according to the size of the tumor, by slight or distinct inflammatory swelling of the immediate surroundings. While from the periphery outward a concentrically progressive new bone formation gains more and more in space, the hemorrhagic fluid is gradually absorbed; the tumor becomes fluctuating and finally doughy. The soft bloody swelling, surrounded by a marginal bony ridge, then appears to the palpating finger like a depression in the roof of the skull. After absorption is complete a slight, uneven periostosis remains for several months. This is the usual course. In larger cephalhematomata with slow subsequent healing there is sometimes the for-



mation of a continuous osseous lamella on the part of the displaced periosteum, corresponding to the vault of the tumor, the condition making itself noticeable by a parchment-like crackling of the surface. The tumor itself and the further process of healing are without effect upon the general condition of the child. Only with very decided extension, as would arise from a varied etiology, would a certain degree of anemia be present. Spontaneous suppuration is exceedingly rare and usually occurs in connection with a simultaneous superficial injury to the skin, which for this reason must be kept particularly clean. In this connection the development of an eczematous disease of the head is serious. Therefore cleanliness with mild prophylactic treatment of the surface with inunctions is the only therapy which appears necessary. Operative removal of the cephalhematoma by division or by puncture with or without aspiration, is strictly contraindicated in uncomplicated cases. Apart from the danger of infection with its serious consequences a secondary hemorrhage and renewed filling, especially at this stage, is not unusual. In the new-born a special pressure bandage cannot be utilized to prevent this on account of the ready compressibility of the skull. At the present time, therefore, the usual treatment of uncomplicated cephalhematoma is expectant. Runge mentions that with an exceedingly large tumor which has existed for weeks puncture with succeeding aspiration may be indicated on account of the danger of bone necrosis, but expressly adds that incision is not absolutely necessary even in these cases until suppuration appears. The latter condition, which may be recognized by reddening of the skin, elevation of temperature and the constitutional disturbance, is the only indication for an immediate operation, which is then best accomplished by broad division.<sup>1</sup>

Associated with external cephalhematoma there may exceptionally be a corresponding *subdural*, therefore an *intracranial*, *extravasation of blood* on the inner surface of the bone of the skull affected. This occurs most frequently after injuries to the bone which have led to a bilateral hemorrhage. Otherwise only congenital split-like defects of ossification are the cause, such as often occur at the points of preference of the cephalhematoma—within the parietal bone and on the tabular part of the occipital bone—and are regarded as typical. The external hemorrhage then simply passes to the inner surface of the skull. Important in the recognition of this complication is the possible presence of cerebral symptoms which are always absent in simple cephalhematoma. These, however, are found only in large subdural effusions of blood and consist of retardation of the pulse, superficial respiration, and sopor, which very soon terminate lethally. The smaller subdural hemorrhages, which are the usual type, generally run their course without symptoms. A further aid in diagnosis, according to the observation of Kirmisson, is the occasional distinct conduction of cerebral pulsation to the external surface of the cephalhematoma which, however, is only possible

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<sup>1</sup> Antiseptic after-treatment is not easily accomplished. In most cases a very small incision or puncture followed by a dry cup (or a Bier suction) is appropriate.—EDITOR.

with broad congenital defects in the bone and particularly in their immediate vicinity.

Much more important to the life of the child than the previously mentioned lesions are *injuries of the cranial bones*. They result primarily from the rapid entrance of the infantile head into the narrow inlet of the pelvis, either spontaneously, as the result of great exertion of the maternal abdominal press, or artificially, by pressure upon the abdomen, extraction at the pelvic extremity, or with the aid of forceps. According to the degree with which the projecting promontory occludes the entrance to the pelvis *groove-like or funnel-shaped depressions arise*. The former, which is the more common of the two, usually involves the anterior border of the parietal bone and lies during labor posteriorly and parallel with the coronal suture. Usually there is only a simple slight indentation, rarely a deep infraction. The prognosis of these changes in themselves is good. Infraction merely affects the external lamella of bone. Larger vessels are not involved. At most there may be a slight external subperiosteal hematoma (cephalhematoma). This deformity becomes dangerous to life, on the one hand, with a simultaneous fracture or splitting of the bone in consequence of the eventual rupture of larger branches of the meningeal artery, on the other by the not infrequent complication of a simultaneous stretching or rupture of the squamous suture, which causes an injury to the transverse sinus.

The *funnel-shaped or spoon-like depressions* are observed most frequently upon the parietal bone, which lies posteriorly during birth, and correspond with the parietal protuberance or the region of the coronal suture; less often they are situated more anteriorly upon the frontal bone. Their greater depth is the reason that the depression of the external lamella has a greater extent and more readily gives rise to the development of a subperiosteal hematoma. With simultaneous penetrating fractures of bone or splinter fractures, especially in the temporal region, this deformity becomes serious on account of the ready injury to the larger vessels. The seriousness of the prognosis is based upon the frequent association of this complication as is proved by the statistical reports of these injuries. One-half of these children are still-born or die soon after birth. In those that survive a direct influence of the osseous impression upon the central organ itself has not been demonstrated. In time a gradual flattening of the funnel-shaped depression takes place. Complete adjustment, however, does not usually occur of itself; on the contrary, even after years a flattening or depression of the affected side of the head can be demonstrated. Of this fact I was able to convince myself in the children of a laborer's wife. The oldest, a boy of seven, showed a distinct lateral flattening of the head. Derangements were not present in this case nor in the other children; the mental activity of the older child particularly left nothing to be desired; he was one of the brightest pupils of his class.

For the treatment of these conditions an operative reposition of the dislocated bones, or, with penetrating fractures and splinter fractures, their



partial removal has been proposed. In that class of cases which, as statistics show, are not only at first but later without symptoms there is absolutely no necessity for such an exceedingly dangerous procedure. Only the presence of cerebral symptoms call for such measures. But even then an improvement in the prognosis can scarcely be expected on account of the slight resistance which even strong and normally developed infants present to operation, and for the reason that in the given case there is in addition a decrease in energy and capability. Nor have I seen any essential success from the employment of an air-pump to raise the dislocated bone. Primarily the difficulty of an entirely air-tight closure of the surroundings! The bell of the air-pump must be manufactured to conform to the shape and size of the head. In the meantime the fate of the child has been decided: Either it has succumbed to a circumscribed intracranial extravasation of blood and the resulting increased pressure of the brain, or it is alive and recovering. In the latter case a further absorption of the hematoma is to be expected. Finally, the cohesion of the fractured bone lamella may require such strength on the part of the air-pump as cannot be considered immaterial to the life of a child in this condition.

*Rupture of the squamous suture*, already mentioned in the discussion of the groove-like osseous depressions, may be observed alone, without this association. According to Fritsch there is a certain amount of tension and loosening of the suture with every difficult extraction at the outlet of the pelvis. As a matter of fact, so long as the skull is retained within the lesser pelvic entrance any decided traction upon the trunk acts in this manner upon that portion of the skull situated below the plane of the pelvic inlet. Thus the extraction of the succeeding head principally predisposes to this injury, much less that of the preceding head by the forceps. Otherwise the scarcely palpable portion of the squamous suture lying between the parietal and temporal bones is greatly broadened. More rarely there is a displacement of the margins of the bones, the parietal bone being forced toward the squamous portion of the temporal bone. The principal danger of this injury lies in a rupture of the transverse sinus, which, as is well known, is situated under the posterior portion of this suture. Aside from the severity of the venous hemorrhage the proximity of the source of hemorrhage to the base of the brain is unfavorable.

Of the lesions at the base of the skull the *separation of the tabular portion of the occipital bone* from its condyloid part is typical. This arises most frequently with extraction of the succeeding head, more rarely with the presenting head, and exceptionally in spontaneous labor. Olshausen regards the transverse compression of the tabular portion of the occipital bone as principally responsible, Fritsch the greater tugging upon the trunk, a partial separation of the suture being caused by the tugging alone or in conjunction with the strong forcing of the tabular portion of the occipital bone underneath the parietal bone, such as takes place from simultaneous narrowing of the inlet of the pelvis in its transverse diameter. The most serious conse-



quence of this separation is the injury to the many contiguous vessels, particularly of the transverse sinus and its continuations. The proximity of the base of the brain and the medulla oblongata increases the danger decidedly. In addition, the loosened tabular portion of the occipital bone may be displaced anteriorly upon the inner surface of the condyloid part and compress the contents of the foramen magnum as well as the portions of the spinal cord and medulla directly above. Although, as Fritsch correctly emphasizes, a direct compression of the spinal cord can scarcely occur since it fills only a small portion of the canal, nevertheless a simple transitory compression may be sufficient for a fatal termination when it occurs with the needful strength such as is contributed by the amount of traction necessary in a constricted pelvis.

In addition to these typical lesions of the skull, difficult birth of the presenting and succeeding head may lead to other manifold injuries: of the bone, from simple fracture to complete destruction; of the sutures, from simple distention to complete separation. The employment of the forceps is often a further occasion of too great compression. The direct effect of this pressure is always localized on the occipital and frontal bones, that of the promontory laterally upon the parietal bones or upon the frontal bone. In extraction at the pelvic extremity the latter arrangement is usual. The more the injury of the bone is located at the height of the skull the less is it important; it becomes the more serious the farther it is below the plane of the parietal protuberance. The principal danger is injury to the vessels and the resulting intracranial hemorrhage. Separation of the sutures invariably results in injury of the sinus, and not only the transverse sinus at the squamous suture but also the longitudinal sinus at the frontal and saggital sutures may be involved.

*Intracranial hemorrhages* have also been observed in spontaneous and even easy and rapid labors without injury to the skull. Here also the chief active factor is compression of the skull during birth, either by the pelvis or by the maternal soft parts. This alone may lead to such sequels with great displacement of the cranial bones. In this manner, in addition to tearing of the subdural vessels rupture of a sinus may occur. As the parietal and frontal bones are especially exposed to great displacement the longitudinal sinus is particularly affected. The superficial, dense mass of tissue sustains the pressure longer, while the weaker wall of the sinus yields. Externally the position of the bones post partum appears normal. Olshausen was the first to call attention to the occurrence of this injury also in spontaneous labor. Naturally such a lesion of the sinus is fatal. In the cranial compression a simultaneous stasis of blood in the interior of the skull constitutes more than an occasional aid. The vessels are incapable of depleting themselves of blood as rapidly as is required by the external pressure. Completely filled they tear more readily. Such intracranial stasis is most frequently the result of asphyxia but may be due to mechanical compression of the vessels of the neck, especially of the veins. Küstner observed intra-

cranial hemorrhages in congenital goiter, and repeatedly when the umbilical cord was wound firmly around the neck. Thus the greater predisposition of premature children is explained. While, on the one hand, the condition which leads to an interruption of pregnancy may produce a disturbance of the intrauterine respiration, on the other hand the skull of the premature infant is softer and more yielding and therefore is readily compressed by a feebler force, the walls of its vessels being less resistant. The seat of these intracranial hemorrhages is usually subdural or subarachnoid and corresponds most frequently to the convexity, more rarely to the base of the brain. Hemorrhage into the substance of the brain is rarely observed. Finally, there are reports of hemorrhage into the ventricles which cause a suspicion of special stasis and compression in the region of the veins of Galen (Kundrath).

In regard to the symptomatology of intracranial hemorrhages, either of spontaneous or traumatic origin, the statement is true that hemorrhages of the convexity within certain limits give rise to decidedly fewer phenomena than those at the base of the brain, and the more so the farther they are situated from the surface. Hence, in conformity with the localization of the hemorrhages occurring spontaneously and for the reason that the lighter grades appear to predominate, as a rule no symptoms are present. When they do arise in consequence of a greater hemorrhage or of a hematoma in the cerebrum they first consist of a retardation of the pulse, superficial respiration, and sopor. For a differentiation from simple asphyxia, and in the absence of external injury to the skull, an occasional more marked prominence of the fontanelles is of value. This observation is all the more important as, with intracranial hemorrhages any great corporeal shock, and therefore Schulze's swings, is contraindicated and a less dangerous and milder method is advisable, for example that proposed by Schroeder. With a basal or massive hemorrhage the soporous condition soon terminates fatally. With smaller hemorrhages in the region of the convexity the severe symptoms may gradually disappear and recovery ensue, or, especially with a subarachnoid or cerebral localization, there may be corresponding focal phenomena: hemiplegia, convulsions. In cases of this kind with hemorrhage at the base of the brain paralyzes of the oculomotor, trochlearis, and abducens nerves appear and occasionally also paralysis of the facial nerve. Their prognosis is at first doubtful, but they tend to recover. That even these have in time improved and finally terminated in recovery is not especially exceptional. As is the case in all intracranial hemorrhages, there is danger of the development of a pachymeningitis which may eventually lead to a renewed hemorrhage (Döhle).

Traumatic injury of the skull and brain *intra partum* is looked upon by psychiatrists as an important etiologic factor in the later development of certain mental and nervous diseases: epilepsy, chorea, athetosis, spastic contractures, spastic paralysis, imbecility, idiocy. In several communications the artificial termination as well as the protracted duration of a labor have been mentioned as having a decided influence upon eventual

serious sequels. Asphyxia also plays an important rôle. In two cases of severe asphyxia B. Schulze noted idiocy later. The course and especially the increase of the condition and its long duration rather favor a simultaneous intracranial hemorrhage. The great percentage of histories of abnormally prolonged labors with operative termination gives cause for much thought. However, the relatives in general are inclined to greatly exaggerate the difficulty of a labor. This number, which is nevertheless small, is opposed by a large series of carefully observed cases in which, even after severe injury, complete recovery and subsequent normal development continued for a number of years. Although it cannot be denied that a later occurrence of these conditions is possible, they must certainly be exceptional.<sup>1</sup>

Among the *nerve lesions* of the head *paresis of the facial nerve* after forceps delivery has been mentioned. This occurs most frequently when the head is grasped obliquely, as the application of the forceps upon or behind the ear presses the trunk of the nerve at its exit from the stylo-mastoid foramen. Such traumatic facial paralysis is not a rare complication after forceps de-

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<sup>1</sup>The author underrates the frequency of the bad results both of asphyxia and of hemorrhages. The latter, besides the causes described in the text, are anatomically predetermined by the insufficient structure of the tissue of the blood vessel walls of the new-born and the shallowness of the impressions digitatae of the inner layer of the skull.

The progress of asphyxia and of its treatment is a very doubtful one in many cases. It depends not only on the knowledge and skill of the physician ("obstetrician"), but on the causes of the abnormal condition. A moderate or serious compression of the head, compression or prolapse of the cord, intrauterine or intravaginal respiration and aspiration of foreign bodies (amniotic liquor, blood, meconium), apoplexy, anemia of the fetus, accumulation of carbonic acid in the blood, poisoning by chloral or morphine taken by the mother, or by her excessive temperature, congenital diseases and malformations, each of them, or several combined, influence both the prognosis of the individual case and the results of therapeutic procedures.

When the long duration of labor, the prolapse of the cord, the protracted compression of the head, the early loss of amniotic liquor, placenta prævia, or a high temperature of the mother endangers the life of the fetus, the best preventive of asphyxia is the artificial termination of parturition. (Comp. Jacobi, *Therap. of Inf. and Childhood*, 3. ed., p. 81.)

Extravasation or thrombosis during a long, or even a brief, asphyxia is the cause of secondary meningitis or encephalitis with its results. Even without meningitis they lead to paralysis, total or partial, idiocy, or epilepsy. The two latter are often combined. Many hundreds of such cases depending on asphyxia have been observed by me, as even the attendants on my clinics in the last fifty years may testify. When I ask: Did the baby cry when born? Did the baby live? The unsuggested reply is that it took minutes, took a long time for the attendant, the midwife, the doctor, to bring the baby to life; that the baby did not behave, did not cry, did not smile, did not notice like other babies, and had slight, or general, or local convulsions. That is why I have impressed upon my hearers the necessity of attending to the baby first, and—unless there be a rare urgency—the mother afterwards.

Intracranial hemorrhages are more dangerous in the newly born than they are even in advanced age in which arteriosclerosis is common. For in the newborn and young infant the blood is less coagulable than later, and the extravasation more copious on that account. That is why so many babies die of intracranial hemorrhage in the first week of their lives; and why every convulsion, and every severe attack of whooping cough in the very young may prove fatal, or—what is more pitiful—the cause of hemiplegia, paralysis, epilepsy, idiocy.—EDITOR.



livery, which appears to occupy a first place among the etiologic factors. This condition is also noted exceptionally after spontaneous labor. Upon extraction of the descending head the peripheral portion of the nerve is not exposed to direct pressure. Marked protrusion of bones in the pelvic inlet, either the promontory or the symphysis, begins higher. Depressions of the bone, directly or indirectly the result of a simultaneous intracranial hemorrhage, may have a damaging effect upon the center of the facial nerve. The immediate proximity of centers for the arm and leg, as well as that of the hypoglossal nerve, makes their involvement, immediate or transitory, unavoidable on account of collateral edema. A damage of the facial nerve center may also occur in spontaneous head presentation through injury to the bone (Schütze). In addition, with a flat pelvis the protracted labor adds to the danger of pressure necrosis of the scalp, and the consecutive swelling in the vicinity, provided it be not too remote from the stylomastoid foramen, may damage the peripheral portion of the facial nerve (Knappe). Another important etiologic factor in spontaneous head presentation in a flat pelvis is furnished by the presence of exostosis of the symphysis. Well-developed anterior parietal bone presentation permits this bony process, similar to the apex of the forceps, to act directly upon the nerve trunk after its exit from the skull. Vogel reports two cases of this kind. In his opinion the observation of Ludwig may be explained in the same manner. This author, however, although uncertain whether the method of development is peripheral or central, believes that the latter is not unlikely. Vogel's contrary opinion that the period of recovery was too short (three to five days) will probably have to be admitted. Finally, there is a fourth report of facial paralysis in anterior parietal presentation with markedly pendulous belly (Frank), which is referred to strong pressure of the shoulders against the aural region. A hematoma of the sternocleidomastoid at its insertion in the skull as a cause of facial paralysis has been mentioned. B. Schulze, to whom we owe this observation, refers the development of the hemorrhage to a strong hyper-rotation of the shoulders after the birth of the head. Recovery occurred in four or five weeks simultaneously with absorption of the hematoma. In the discussion of this report Olshausen pointed to the influence of amniotic strands in the sense of a tugging at the affected area. Geyl thus attempts to explain a case observed by him, without, however, giving positive objective reasons.

Corresponding to the usual nature and the place of development *paresis of the facial nerve* is unilateral in by far the majority of cases, and shows itself clinically in a disturbance of function of the facial muscles supplied by it which is especially obvious when the infant cries. The affected side remains flaccid and immotile; the healthy side, in contrast, shows a marked deviation. The mouth and nose are drawn toward the normal side. The nasolabial fold, more prominent on the unaffected side, has disappeared upon the paralyzed side. The eye upon the affected side remains open (lagophthalmus). Usually the entire trunk of the *pes anserinus major* is affected, more rarely only a portion and then the lower, which supplies the nose and

mouth. The *prognosis* of this unilateral peripheral facial paralysis is always favorable. As a rule the affection disappears within a few days. This rapid improvement sometimes refers only to the upper branches (eyes) while recovery of the branches of the mouth takes place more slowly and is not completed before two or four weeks. In a few cases entire lack of improvement and persistence in later life has been noted (Duchenne, Henoch, Selig-müller, etc.). This naturally presupposes a severe labor with total destruction of the nerve. In the differentiation of this purely peripheral nerve lesion beyond the stylomastoid foramen from the intracranial or basal form the condition of the superficial petrossal nerve, that is, the velum of the palate, upon the affected side is important. It is true, the normal condition of the latter will be a more positive warrant that the lesion has occurred peripherally from this branch. The inverse condition we will rarely have opportunity to demonstrate in the new-born. Basal facial paralysis is usually due to a basal injury of bone and intracranial hemorrhage, which on account of their position and their contiguity to the medulla oblongata at once give rise to severe cerebral symptoms.<sup>1</sup> The same is true of bilateral facial paralysis, singly or with involvement of the ocular muscles. The limitation to the nerve alone is scarcely likely on account of the smallness of the space. That for this reason the prognosis of these intracranial nerve lesions usually offers but little hope of recovery need scarcely be remarked. The estimation of injuries of the facial nerve center is according to the same fundamental laws as have already been detailed of cerebral lesions in general. Simple peripheral nerve paralysis alone is influenced by treatment. This, however, is rarely necessary on account of the usually rapid spontaneous cure. Only in those cases which show no improvement in the second week is faradization indicated. For similar reasons the prolongation of the lagophthalmus exceptionally may require corresponding protective measures for the eye. Finally, a hindrance of nourishment through difficulty in sucking is possible. This only occurs with a severe disturbance, and then when the child is fed at the breast. I have never come across a case of this kind. Under such circumstances the bottle will be a serviceable substitute.

*Other injuries to the face* are rare. In face presentation a careless and rough examination may be an accidental cause. Otherwise pressure of the forceps is the most important factor. In a narrow pelvis the forceps applied to the head, which is still high, occupies the transverse diameter. One blade of the instrument lies more or less across the face. In this way the nose may be injured and *fracture of the nasal bone* may result. Olshausen saw this injury after a spontaneous labor. The possible *lesion of the upper eyelid* by the forceps has already been mentioned. Prophylactically, it is advisable even in a severe labor to avoid too great compression of the handle of the forceps. In the Mauriceau or Veit-Smellie maneuver the *mouth and oral cavity* are the points of attack. The introduction of two fingers may tear the angle of the mouth; great pressure upon the floor of the mouth

<sup>1</sup> See "Diseases of the Nervous System," p. 674, *et seq.*



may cause suggillation, compression, or a lesion of the soft parts situated there; finally, too strong tugging upon the lower jaw may cause its fracture or complete separation. Therefore, careful management and manipulation of this method in the living child is absolutely necessary.

In the *neck* superficial injuries of the skin upon the anterior side in the form of transverse *distention streaks* are familiar in face presentation. They are due to great tension of the skin. Kaltenbach also saw these lesions upon the nape of the neck in natural occiput presentations. Fat children appear to be particularly predisposed to the latter. Recovery occurs spontaneously within a few days. When the injuries are of decided extent a treatment with salves may be necessary. *Deeper injuries to the soft parts* occur principally under the typical picture of a subcutaneous hematoma on account of the predominant torsion of the muscles. They have been observed after artificial as well as spontaneous head and breech presentations and occur most commonly in the *sternocleidomastoid muscle*, *more rarely in the trapezius or the scaleni*, and exceptionally in the latter muscles alone. The hematoma is situated laterally and, according to the region and cause of its development, near the head or in the vicinity of the shoulder. As a rule it is unilateral, but a bilateral development has been observed. After a spontaneous labor, as a rule, its extent is slight, not greater than the size of a cherry; the more marked grades are usually the result of operative termination of the labor. We owe to Küstner the investigation and proof of the various origins of these injuries as well as the active mechanism, particularly as by this means we have obtained important indication for prophylaxis when operation is necessary. According to this author a small number owe their development to direct force. This may occur with the forceps by the greater pressure of one blade, as well as in a breech presentation by extraction with the finger placed over the nape of the neck and laterally to the neck. In the majority of cases the injury is caused indirectly by immoderate distention. This may happen to the trapezius muscle and to the scaleni by tugging in the longitudinal axis of the child. The same lesion to the sternocleidomastoid is possible by extreme rotation of the head, whereby the inactive muscle, on the side to which the face is directed, is stretched. The isolated hematoma of the sternocleidomastoid is most distinct after spontaneous delivery, when there has been no disturbance from tugging in the longitudinal axis. Here the retention of the shoulders is especially serious as the head, which is at the exit of the pelvis, lies with its breadth in the broad pelvic diameter. A similar inverse mechanism may occur in spontaneous breech presentation. If, in spontaneous delivery, the active natural effort is sufficient for the laceration of a muscle, as Küstner's investigations indicate, such an accident is all the more likely in an artificial rotation of the head by the forceps, or of the shoulders in a breech presentation. If in this rotation the tugging occurs in the longitudinal axis of the child the injury correspondingly may show a wider distribution to the splenii and to the trapezius muscles. The prognosis of this injury to the soft parts (as regards life) is always good, and is generally favorable as regards func-



tion. According to Küstner even large hematomata disappear spontaneously in four to eight weeks. The same is also true of paresis of the muscles of the shoulder and arm which is sometimes present simultaneously and for which Fritsch considers the direct pressure on the nerve by the finger of the obstetrician or the compression by the hemorrhage as responsible. Strohmeyer's view that tearing of the sternocleidomastoid readily produces a myogenic contracture—a *caput obstipum*—has been corrected by Peterson; accordingly we must regard this sequel as at most very exceptional. Small and even medium-sized hematomata heal without leaving a trace. Only with a large effusion of blood may it be advisable to prevent a possible residuum by the appliance of a proper bandage and afterwards by mild massage and passive movements.

Extensive injuries to the soft parts of the neck are usually combined with injuries to vital organs, especially of the skull and brain. When these are absent the simultaneous nerve injury makes a return of movement very questionable. Paralysis of this origin are characterized by atrophy of the muscular area which develops within a few days.

The employment of extreme force at the shoulders in a breech presentation or on the descending head in head presentation may also endanger the *vertebral column*. This structure is well protected by its powerful ligamentous apparatus to sustain an energetic tugging so long as this occurs in the direction of its longitudinal axis. Deviation to one or both sides may be serious. Epiphyseal rupture, hemorrhage into the spinal cord canal, tearing or separation of the spinal cord or of the nerves originating here are easily the consequences. Whether hemorrhages may occur in the spinal cord canal after delivery by simple tension, without direct injury to the bone, has not yet been proven. Practical experience is opposed to this, according to which later disturbances are exceptional. Neuropathologists, however, have recently taken the view that they play an important rôle in the etiology of subsequent diseases of the spinal cord.

In the region of the thorax *injuries to the lungs* may occur, as in adults, from fractured ribs and exceptionally from a fractured clavicle. But even without such injury hemorrhage into the pleural cavity has been noted. The cause was then usually based on the tearing of over-full venous nets of many branches, situated upon the anterior side of the vertebral column, particularly in asphyxiated new-born.

The *organs of the abdominal cavity* are especially exposed to damage by an improper or unskillful delivery in breech presentations: when the abdomen is grasped firmly and roughly either in extraction or in raising the trunk to free the arms. Such injuries are also reported in difficult labor associated with great tugging and distention of the trunk: rupture of the liver or spleen, hematoma of the adrenals, fatal hemorrhage into the abdominal cavity. In view of the fact that in these cases asphyxia was present it is uncertain how many of these sequels were due to this cause.

*Birth trauma of the extremities* results in injuries to the bone (infraction,

fracture, epiphyseal separation) and paralysis of the nerves, alone or in association. Before entering upon a description of the bone lesions it may be advisable to devote a few words to the *so-called intrauterine fractures*. Until recently certain single angular bendings of the tubular bones, presenting great similarity to dislocation, healed fractures, and infractions, were thus incorrectly designated. They are observed most commonly in the lower leg, rarely in the forearm, and exceptionally in the clavicle. Bony deposits or thickenings in the vicinity of the inflexion were regarded as residues of an excessive callus formation, and slight cicatricial cutaneous changes in this region as injuries of the skin caused by the fractured ends. This conception of an intrauterine origin of fracture of bone, which occasionally was supported by the statement of the mother of a trauma during pregnancy, was a more welcome and unobjectionable explanation. Where such an accident was not admitted no question was raised, as the power of the contracting uterus during pregnancy was considered responsible. Recent investigations (Max Sperling, Haudeck, Burmeister, etc.) have shown that these deformities are not the consequence of an intrauterine traumatic bone injury; on the contrary, they are simple disturbances of development. In the preparations examined there was no trace, macroscopically or microscopically, of a total or partial interruption of continuity. In the axis the bony substance was unchanged and uninterrupted. Nothing in the nature of a fracture cicatrix or of a change which could be regarded as such could be seen. The periosteal covering also, except for a slight annular distribution of its numerous cells corresponding to the point of inflexion, was unaltered. The osseous thickening, regarded as callus formation, proved to be a simple, osteoplastic, subperiosteal callus which was usually more strongly developed on the concave side of the inflexion and here caused a certain compensation of the angle. Finally, the skin was entirely normal with the exception of the cicatricial retractions which showed a flattening of the corium papillæ and a thickening of the lower layers of the epidermis. No signs of cicatricial tissue were ever present. If the view of a simple inflexion and curvature of the tubular bones without any interruption of continuity was considered very likely at that time it has recently received confirmation by the Roentgen pictures of these deformities. The contour of the bones is perfectly uniform; irregularities such as occur in the healing of dislocated tubular bones from an immoderate formation of callus are not observed. Marked angular inflexion of the tubular bones without interruption of continuity can only occur at a very early period of fetal life, before ossification has begun, or at least before it is much advanced, therefore, according to embryologic investigations, within the first two months. The defective development observed in 60 per cent. of the cases makes it obvious that the beginning is prior to the completion or internal differentiation of the stump of the extremity. As a casual factor external trauma, notwithstanding the necessarily associated sequels (injury to the maternal soft parts, interruption of the pregnancy) must be rejected on account of its transitory effect. The same is also true

of contractions of the uterus. Only the influence of prolonged limitation of space can be considered, either as it concerns the fetus as a whole (deficiency of amniotic fluid) or the restriction of individual portions of the body by amniotic strands and folds. A few authors regard tumors of the uterus (myomata), according to their situation, as responsible. The comparatively great frequency of an associated defective development makes it probable that the influence of amniotic strands is an important and common cause of these deformities also. Positive supportive criteria are furnished by the cicatricial cutaneous changes at the point of inflexion and remains of strands in the placenta or upon the affected extremity. This is also the preferred explanation of the origin of periosteal osseous thickenings or of the localized periostitis which is the foundation of them, as well as of the defective development of the portion of the extremity situated peripherally from the point of inflexion.

After excluding these solitary angular curvatures from the various *spontaneous intrauterine fractures* there still remain the exceptional occurrences of ununited fractures of bones. Besides the atypical localization their multiple occurrence in the same child is characteristic. Complete interruption of continuity is rare; usually there is only an infraction. The causes of this slight capability of resistance and ready tendency to break have been found macroscopically to be an abnormal formation of bone substance, deficient and irregular deposits of calcium, extraordinary increase and dilatation of the Haversian canals, and substitution of the spongiosa by a rich-celled connective tissue. These changes were formerly regarded as similar to those of true rickets, with which, however, the normal composition of the bony boundary of the epiphysis does not agree. The fundamental cause of this bone disease is still unknown. A general nutritive disturbance of the child or of the mother is usually invoked in explanation. According to some authors previous syphilis plays a decided rôle. It must also be remembered that specific luetic processes within the tubular bones result in a similar tendency to fracture. Naturally, such spontaneous fractures of bone occur most readily at birth, favored by a special position of the affected member. A few reports indicate an intrauterine origin. Their direct cause, whether from contraction of the uterus, from energetic movements of the child, etc., is entirely within the realm of hypothesis.

With a normal physical development injuries of the bones of the extremities are due principally to an operative termination of birth and are particularly common in extraction in breech presentation when the course is altogether abnormal or when obstinate conditions exist, such as contraction of the pelvis, resistance of the maternal soft parts, etc. Here also the regularity of the active factors creates a certain uniformity of the injuries and entirely justifies the designation of typical. In the upper extremity and its shoulder girdle *fracture of the clavicle* is most common. This may occur during the release of an arm and also in head presentation. Great limitation of space occasionally renders the first introduction of the hand serious, in



that the shoulder and clavicle of the child are immoderately compressed in their longitudinal diameter. In other cases this result during the release of the arm occurs through the agency of the upper arm, analogous to the production of fracture of the clavicle in the adult from a fall upon the extended limb. More rare than this indirect mechanism is a direct cause, a longitudinal compression having occurred by too great retraction of the shoulders in the maternal pelvis prior to the release of the arm, or in head presentation by the pressure of the finger upon the shoulder. According to Küstner too great pressure upon the shoulder as well as forced traction upon the arm may also exceptionally cause an epiphyseal separation of the sternal end of the clavicle. As a rule the ligamentous apparatus here offers such resistance that fracture of the bone takes place instead. The fracture of the clavicle, which is similar to the infractions of adult life, is usually at the boundary of the external and middle third of the bone, rarely in the middle. As a dislocation of the fractured ends is not usual the diagnosis is generally based upon the results of palpation: abnormal inflexion in the area concerned, decided pliability upon slight pressure, crepitation. Lower position of the affected shoulder and visible decrease in breadth of the corresponding half of the shoulder are only perceptible with marked deviation. Unless careful investigation is made the injury often remains undiscovered until the beginning of the second week, or until a perceptible callus formation directs attention to the condition. Besides the scientific interest and the consideration for the child it is always advisable for personal reasons for the obstetrician after every labor to examine the child minutely for injuries and thus avoid unpleasant surprises and the resulting criticism on the part of the relatives.

The prognosis of fracture of the clavicle is always favorable. Even if undiscovered and untreated it heals [mostly.—Editor] without sequels. Special treatment of simple infraction is quite unnecessary. With complete separation and consecutive dislocation of the fractured ends a light fixation of the arm to the wall of the thorax—which is protected by cotton—by means of a few turns of the bandage or of a triangular cloth is sufficient. The bath need not be denied the child if, as Fritsch advises, the bandage is not removed and is subsequently renewed. Especially anxious mothers may omit the bath and leave the bandage on until the end of the first week, when the callus which is usually palpable indicates the beginning of consolidation. In these cases the skin should be carefully anointed to prevent a possible development of eczema, and for the same reason a wetting of the bandage makes an immediate change imperative.

Much more rare than these clavicular lesions are *fractures of the humerus*. This bone is particularly endangered in releasing the arm, especially when that member is turned upward or is bent behind the neck, and above all when it is lodged between the head and the promontory or symphysis. The most common accident is *fracture of the diaphysis and epiphyseal separation* near the head of the humerus. The former presupposes that the force of the trauma has occurred near the middle of the diaphysis. Besides the amount

of strength used the smallness of the point of attack plays an essential rôle. The release of the arm by one finger is therefore more dangerous than if several fingers were employed. Usually the first symptom that is conspicuous is a hindrance of function. The injured arm remains immotile. The fingers alone, particularly their flexion, are less implicated. Diagnostically, in addition to the greater passive movability at the abnormal area the crepitation which is perceptible here is conclusive. Furthermore the child always screams when the affected extremity is moved and particularly the ends of the fracture. Formerly it was necessary to depend alone upon these objective phenomena and because of the smallness of the diaphysis errors were quite common; the last few years, however, have furnished an absolutely positive criterion in the X-ray picture. This should be utilized not only in doubtful but also in perfectly obvious cases since by this means the nature and position of the fracture can be accurately determined.

The prognosis of fracture of the humerus in the new-born cannot be regarded as unfavorable. Although it is not always possible entirely to exclude deformity, the resulting disturbances are exceedingly rare. As to treatment, the reposition is usually easy but the retention exceedingly difficult. A bandage too tightly applied may be the cause of muscular atrophy. A loose bandage has no influence upon the dislocation. It is difficult to find the correct alternative between these two extremes. Fritsch merely ties the upper arm at a right angle with the thorax and has this bandage renewed after every bath. The moderate mobility of the fingers and hand which remains prevents later contractures. A dislocation even then cannot be altogether avoided but the deleterious results, according to practical experience, are slight. Although every practitioner must regard this method of Fritsch as correct, nevertheless he will do well, in the beginning at least, to omit the daily change of the bandage and the bath unless he desires to avoid the reproach of the relatives for negligence and other unpleasantness. However, the bandage must be controlled and renewed in three or four days at the latest and may be serviceably associated with the bath. It is also advisable to cover the upper arm as well as the thorax with a thin layer of cotton and previously to anoint the skin. In the application of bandages around the thorax it must always be remembered that the chest of the new-born is readily compressible and that very little pressure is required to constrict and hinder the respiration.

In addition to the fixation bandage retention of the fractured ends may be accomplished by permanent extension. This treatment, and especially the method of Bardenheuer (Cologne) which has been tested in many cases, has given the best results, especially in fractures of early infancy. By pulling and opposed tugging the fragments may be easily and securely retained in their normal position. All atrophy from inactivity or pressure of the muscles is excluded. The skin of the newly born infant bears the necessary adhesive plaster bandage very well. Above all, with this method the nursing and care of the child are absolutely unhindered. The daily bath had better be omitted, but an occasional bath may be given notwithstanding the bandage



as the subsequent readjustment of the entire apparatus is very much easier. Briefly, the employment of permanent extension in the cases in which dislocation occurs readily is to-day considered of particular advantage and furnishes the best results.

*Epiphyseal separation* may occur in a manner similar to fracture of the diaphysis if the point of greatest force upon the humerus is close to its boundary of ossification. The best example is the hooking in the axillary cavity after the development of the head—during extraction of the trunk—when the arms are still placed laterally to the body. Also serious in this connection are all movements of the arm which appear to be opposed to the normal structure of the shoulder-joint: rotation about its longitudinal axis, movement of the arm over the back of the child and near the body. Küstner has pointed out that the flat, only slightly concave formation of this marginal plane in the new-born decidedly favors the occurrence of injury. For a proper understanding of the sequels it must be remembered that in the new-born the epiphyseal boundary is external to the capsule of the joint and therefore, in such a separation, this structure may remain uninvolved. As in diaphyseal fracture, a hindrance of spontaneous movement first calls attention to the condition. Furthermore, the affected arm assumes a very characteristic, almost pathognomonic position, varying according as to whether there is merely a loosening of the epiphysis without injury of the periosteum or whether the latter structure also is injured and the diaphyseal end correspondingly dislocated. In the former case the damaged extremity shows a strong inward rotation of the upper arm, perhaps associated with hyperpronation of the forearm. In the new-born all of the outward rotators are inserted at the epiphysis, their antagonists, with one exception (subscapularis), in the shaft of the diaphysis. With the separation of the bone the antagonistic action of both muscle-groups is destroyed and each may act independently of the other. The action of the outward rotators is limited to the epiphysis and remains concealed from the eye of the observer; that of the inward rotators comes into the position of the diaphysis and of the lower epiphysis united to it. The periosteum tears upon that side to which, on account of the force, the diaphyseal end has deviated. If the end slips back at once the condition is the same as in simple separation of the epiphysis without injury to the periosteum, therefore merely an inward rotation of the upper arm. If, however, the diaphysis remains dislocated the upper arm assumes a position similar to that of luxation. If the periosteal tear and dislocation affects the axillary side the longitudinal axis of the upper arm, as in an axillary and subcoracoid dislocation, deviates anteriorly and downward from the articular plane. If this condition has occurred on the external side the deviation is posterior. As the head of the joint in epiphyseal separation remains in the plane the corresponding flattening of the shoulder is absent and the curvature of the diseased member is exactly like that of the normal. Further, in contrast to the objectively distinct, limited movability of the shoulder-joint after a dislocation, this movement appears to be par-



ticularly facilitated after an epiphyseal separation. Finally, traumatic luxation of the shoulder-joint in the new-born is absolutely impossible under normal circumstances on account of the slight resistive power of the epiphyseal boundary. Only in very exceptional cases where the union of the joint has undergone a disturbance of development, or ossification of the diaphyseal and epiphyseal boundary has occurred intrauterine is such a condition possible. According to Küstner's careful analysis those cases of traumatic luxation in the new-born which are found among the earlier reports are erroneous.

Diaphyseal rupture differs from epiphyseal tearing not only in the pathognomonic position but by the absence of distinctly palpable rough crepitation. As the separation occurs in the cartilaginous layer a soft, so-called cartilaginous, friction at most is noticeable. Naturally, in addition to the tear of the periosteum there may also be injury of the adjacent soft parts. This, however, is usually slight; large effusions of blood are rare. Involvement of the nerves is more common. Corresponding to the anatomical relations of this region a dislocation of the diaphyseal end toward the axillary cavity is chiefly the cause of this condition. Not only the nearest nerve, the axillary, is endangered, but also the entire lower portion of the brachial plexus which innervates the arm. The condition is usually that of compression, rarely of tear. Eventual sequels are at first concealed by the general immovability of the arm brought about by the trauma. Paralysis and muscular atrophy do not arise until after consolidation has taken place.

In the special diagnosis of epiphyseal separation in the new-born these clinical symptoms are of principal importance as the X-rays are of much less service here than in fracture of the diaphysis. The epiphyses are still cartilaginous and are penetrated by the rays the same as other soft parts and therefore cannot be distinguished skiagraphically. Nevertheless, an important confirmation of the clinical findings may be indirectly obtained by exclusion.

The *prognosis* as regards the use of the affected arm, aside from a possible nerve paralysis, even after the correction of the dislocation is essentially dependent upon the careful prevention of an opposed rotation of the two fractured ends. If the latter condition is overlooked the epiphyses, rotated outwardly, will heal upon the reversely turned diaphysis and the movements of the arm will be much restricted or entirely impossible. Therapeutically, therefore, in a dislocated epiphyseal separation the correction of the *deviation ad axin* comes into question and can usually be brought about by simple traction of the arm. The second requirement, the relief of the dislocation at the periphery, can only be accomplished by bringing the diaphysis into a position which corresponds to the shorter end—a direct influencing of the epiphysis, which is inaccessible. With this outward rotation, even if it occurs somewhat more posteriorly, the upper arm can only be fixed uncertainly and with difficulty to the thorax of the new-born. Küstner therefore advises the employment of the forearm as a second splint: The upper arm, after rotation outward, is fastened to the strongly supinated forearm, which is flexed at an

acute angle so that the hand lies upon the injured shoulder, and both are fixed to the thorax with an axillary pillow between. Preferable to this very difficult process is the employment of permanent extension, from which, (according to the reports of Jetter, Wolff, Linser, and others) in the treatment of this injury particularly, the most positive results are obtained. Operative procedure—direct fixation of the epiphysis to the diaphysis by a steel punch (Helferich) which until recently was regarded as the only sure method—can only come under consideration when simple reposition is prevented by compression of soft parts (button-hole tear in the periosteum), which in the new-born is exceedingly unusual.

*Other injuries of bones of the shoulder-girdle*—epiphyseal separation at the collum scapulae, separation of the acromion, transverse fracture of the scapula, and epiphyseal tear at the lower end of the humerus—are so rare that their discussion may be omitted.

Thus far only such *paralyses of the arm* have been considered as develop in consequence of central lesions of the brain and of the spinal cord or immediately after injuries to the bone. Cerebral localization requires, apart from a simultaneous disturbance of the general condition, a more or less distinct involvement of other neighboring focal areas and affects at least the entire upper extremity just as in the myelogenous form. In contrast thereto, in a peripheral secondary injury to the brachial plexus, on the one hand, the paralysis is incomplete, that is, certain parts, usually the hands and fingers, are still capable of motion; on the other hand, it is also combined, as several nerve trunks of this plexus are functionless. The special composition is dependent, in addition to the direction and extent of the active power, upon the region of the primary bone injury. The higher this is situated the more nerve trunks are involved; otherwise a few may have branched off previously and therefore remain intact. This criterion applies particularly to the difference between an epiphyseal separation and a fracture of the diaphysis. Fracture of the clavicle is apparently an exception. Notwithstanding the fact that it is comparatively the most common, complicating paralysis of the arm is very rare. This, however, is readily comprehensible when we consider that the effect of the force is here usually not vertical but is directed parallel to the body and is also exerted downward, especially in releasing the arm.

In addition to this indirect, secondary damage to the nerves there are also *direct, substantive injuries*. Their first comprehensive description we owe to Duchenne; a minute knowledge of their localization to Erb. The nature of the damage and how it occurs intra partum is yet a matter of controversy. *Erb-Duchenne paralysis*<sup>1</sup>, as this variety is called, reveals itself in paralysis of the deltoid muscle, the biceps, brachialis internus, infra-spinatus, teres minor, and the supinators, and therefore includes the regions of the axillary, supra-scapularis, musculo-cutaneous, and radial nerves. It is evident that the lesion must have affected the plexus in an area in which

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<sup>1</sup> See volume on "Diseases of the Nervous System," p. 720.

these nerve fibers lie close together, before they have branched, and thus, in the course of his investigations, Erb succeeded in finding a point at which, by means of the faradic current and the use of small electrodes, a common energetic contraction of these muscles could be produced. This region, known as Erb's point, is situated at the place of exit of the sixth cervical nerve, between the *scaleni* muscles. He referred the seat of the lesion to this point and the roots situated centrally from the plexus, therefore to the fifth and sixth cervical nerves, which has been confirmed by the subsequent investigations of other authors. The clinical picture of this form of paralysis is described by Erb and by Duchenne as very characteristic: "The affected arm droops immotile to the side of the trunk; it is rotated inward and permanently extended; neither flexion of the forearm nor raising of the arm is possible; on the other hand movements of the fingers and of the hand are retained." According to both authors the very rapid degeneration reaction is conspicuous. In addition to this there is another clinical symptom—a contracture of the non-paralyzed muscles. The prognosis, according to Erb and Duchenne, is not invariably favorable; neglected cases sometimes do not recover. Although Duchenne advises early faradic treatment he believes that the same results can be obtained by the galvanic current. Erb's teaching is still correct; the only change that has arisen relates to the causal development of this form of paralysis. His investigations were made principally in adults. The functional disturbance, it is true, had existed from birth, but as to the course of the labor and the required operation he was necessarily dependent upon the anamnesis alone. As these always showed an operative termination (version and subsequent extraction, or traction at the shoulders to aid the trunk) he believed that, in head presentation especially, the traction of the shoulder with the index finger introduced in the form of a hook into the axillary cavity, and in version and extraction the difficult release of the arm and the energetic employment of the Prague method were at fault. The same factors, confirming Erb's opinion, are mentioned in the subsequent publications of other neuropathologists. Hoedemaker and F. Schultze add that in raising the shoulder and the arm, especially when the latter is directed upward or under the nape of the neck, the middle portion of the clavicle is approximated to the transverse processes of the seventh and eighth cervical vertebrae and thus compression of the fifth and six cervical nerves becomes possible. The greater the curvature of the clavicle and the less the amount of fat, the more likely is this pressure paralysis. The character of all of the investigated material was the same: the cases were always observed later, never during the course of the birth. That obstetricians were skeptical regarding this form of paralysis is little to be wondered. The similarity of the causal factors with those of injuries to the bones, the coincidence of the clinical symptoms, particularly the position of the arm, the unfavorable prognosis, and finally the difficulty in diagnosis, especially in epiphyseal separation, warranted the suspicion that in the cases in question there was an injury to the bone which had not been recognized. As lately as 1880



Küstner mentioned this view in his monograph upon Injuries of the New-born. The labors of the last decade, however, have shown this opinion to be altogether erroneous. Aside from the fact that even obstetrically a number of positive cases of pure Erb-Duchenne paralysis have been reported, which in the last few years have also been positively confirmed by means of the X-rays, many investigators have studied the origin of this nerve lesion experimentally. An investigation from the present stand of this theory of development is all the more advisable since it furnishes exceedingly important criteria for an eventual prophylaxis. Erb-Duchenne paralysis has been observed after spontaneous head and breech presentations as well as after those of operative termination. In the former the children were always comparatively large with especial breadth of shoulders. While the birth of the head occurred unaided or only with slight assistance, the delivery of the shoulders was difficult, as they remained quite or almost within the transverse diameter of the pelvis or else the anterior portion was lodged behind the symphysis. The positive and only means of facilitating the labor is slight traction of the head. So long as this remains in the direction of the body axis the tension upon each plexus is the same, and unless there is too great force no damage results. The situation becomes critical only when the head is inclined toward one shoulder, to which the mechanism of the delivery of the shoulder is readily prone. If the tension is concentrated to one side of the neck alone a simultaneous traction readily increases it to actual tugging or even tearing. Fieux, and subsequently Schoemaker and Stolper, have tested this method of development experimentally. According to them the fifth cervical nerve is not only first but most involved, the sixth later and to a less extent, while the seventh and eighth cervical nerves remain entirely unaffected. This division confirms the practical observation that the deltoid, the nerve of which proceeds principally from the fifth cervical nerve, may be paralyzed alone, and in the simultaneous development of other muscle areas its paralysis appears to be the most marked and most tenacious. This tugging upon the laterally inclined head in the deliveries that are otherwise spontaneous is the most common and probably also the most readily understood cause of development of Erb-Duchenne paralysis. According to the findings of Fieux and Schoemaker, when the anterior shoulder is compressed behind the symphysis the weight of the head alone may be sufficient, provided there is no support, therefore when it hangs over the perineum. This factor is of special significance in this form of paralysis occurring after an absolutely spontaneous delivery, the statement usually being made that the child was very large and the trunk followed slowly. In a case of this kind Polaillon believed a compression of the plexus by the clavicle, which had occurred previously intra partum, to be due to a too great compression of the shoulders in the bony pelvis. In head presentation, however, under these conditions, the clavicle is simultaneously forced toward the feet of the child and thus the possibility of compression of the plexus is very slight if not altogether improbable. The approximation of the clavicle to the vertebral column and the first rib and

a possible compression of the plexus at this point only becomes active in head presentation when the finger is hooked in the axillary cavity, either incorrectly from the side of the abdomen or when the back of the finger is introduced too far and its point during traction presses directly upon the clavicle. Finally, in spontaneous head presentation the eventual effect of exostoses of the pelvis much be remembered. Just as with the facial nerve at its point of exit from the stylomastoid foramen (Vogel) may Erb's point also be exposed thereby to pressure, which appears to be decidedly facilitated by deflection of the head, especially in a face presentation.

Examples of this form of paralysis after the use of forceps are reported by Danyau, Depaul, Guéniot, Thorburn, Bollenhagen, and others. As proof of the effect of direct pressure of the apices upon the plexus the finding of the remains of pressure or distinct pressure marks in the region of *Erb's point* may be mentioned. That such a direct pressure effect is possible anyone may convince himself upon the manikin. The greatest danger lies in deflection of the head, therefore in anterior head, frontal, and face presentations, and becomes the more extreme the greater the grasp is lowered when locking the forceps and with the forceps applied in a vertical position. Naturally, in addition to this there is the danger of too great compression of the blades. According to Schoemaker and Stolper the rotation of the head by the forceps without a simultaneous tug has proved serious. Here the damage does not occur merely from simple compression toward the cervical vertebral column; on the contrary, the yielding of the muscles of the neck permits a simultaneous tension and torsion of the nerve trunks. This perhaps may explain the more serious prognosis of this form of paralysis than of paralysis of the facial nerve from forceps compression. On account of the comparatively broad attacking surface of the apex of the forceps the damage is not limited to the nerve alone and lesions of the surrounding area such as suggillations or large effusions of blood are not unusual and enhance and protract the injury to the nerve trunk.

Cases of Erb-Duchenne paralysis after spontaneous labor and after head presentations with the aid of the forceps are very rare. The majority of those which have been reported have followed breech presentation. The most serious procedure is the release of the arms. From the experimental investigations of Schoemaker and Stolper it appears that upon raising the shoulder the clavicle primarily approximates the first rib; strong upward pressure with simultaneous compression toward the median line forces it against the vertebral column. Such a consequence arises when the arm is directed upward or under the nape of the neck, therefore with considerable shoulder breadth and narrow pelvis. The plexus is then liable to a compression either between the clavicle and first rib or between the clavicle and the vertebral column. In the cadaver this effect of raising the shoulder is decidedly increased by simultaneous pressure from behind; in practice it is more rare, as the release of the arms usually occurs from the back. Improper rotation of the trunk and release of the arm from the abdominal side

of the child may perhaps bring about such a result. Notwithstanding the former opinion of Erb the plexus appears to be least endangered in the Mauriceau and the Prague methods. Here also, naturally, an inclination of the head, therefore deviation of the force from the axis of the body, is serious. Otherwise the fingers only meet Erb's point by strong curvature of the terminal phalanges and injure it the more they bore into the soft parts of the neck. In the main the Prague method has been rejected and is no longer in use.

In accordance with this method of development we may contrast a form of paralysis due to compression of the plexus with that resulting from too strong traction. The prognosis of the former appears to be decidedly more favorable. In certain cases the outlook appears to depend exclusively upon the result of accurate faradic and galvanic tests of the existing irritability of the muscles and nerves—upon the reaction of degeneration. In regard to treatment, most authors believe the early institution of electric treatment to be essentially important. Nevertheless, months not infrequently elapse before an obvious result is attained.

In the *lower extremity* the most serious accidents are *superficial pressure ulcers* or occasionally deep compressions or even wounds at the flexure of the thighs after difficult delivery in breech presentation, either with the finger or with the aid of instruments, hooks, or slings. All other lesions, particularly of the bones, are the result of a faulty method of version or extraction. The deviation of the fingers, hooks, or the sling from the flexure of the thighs against the upper thigh causes a fracture within the upper third. This misfortune has also occurred in version, due either to the crossing of the legs or to the attempt of the obstetrician to act from the beginning more upon the thigh than upon the foot. The diagnosis of this bone injury is easy: disturbance of function, crepitation, abnormal passive motility, and the findings of the X-rays. Epiphyseal separation of the femur and of the bones of the lower leg is due to incorrect turning of the extremity during traction. Therapeutically, in all of these cases, as in adults, accurate replacement is the first consideration. The later retention is most readily accomplished by the employment of permanent extension with vertical elevation. Less serviceable is the proposal of Credé to fix the extremity upon the trunk, as by this means the dislocation is not sufficiently removed.

The occurrence of *luxations* is the same as in the upper extremity. These are throughout merely congenital anomalies of development.

In the lower extremity there is little opportunity for the development of paralysis from tension of the nerve trunks as the buttocks find no resistance upon their entrance into the pelvis. Nerve lesions are rare even in injury to the bone as here the abnormality of movement is of more influence than the greater employment of force. There remain, therefore, only paralysees of central origin, cerebral or myelogenous. The former are observed after difficult forceps delivery with contracted pelvis, are always



unilateral, and usually involve the corresponding arm and the facial nerve. The disturbance of function within the lumbar cord is the consequence of difficult extraction in breech presentation, with stretching of the vertebral column so that the spinal cord itself tears or hemorrhage appears in its enveloping membranes. In either case paralysis of both legs occurs and if no other injury is present in the upper portion of the body—the cervical cord and brain—it is limited to this region alone.

## DISEASES OF THE UMBILICUS

After the birth of the child the first indication is *ligature and severing of the umbilical cord*. If no contraindication, such as asphyxia, is present which requires some other method it is customary to wait until the reserve blood in the placenta and in the vessels of the umbilical cord has entered the body of the child. This method was first introduced at the beginning of the last century by Messmer and Ziermann and was later recommended by Budin and Schücking. Although the reasoning of these authors has been shown by subsequent investigations (Eröss, Schiff) to be much exaggerated the method is nevertheless of some importance within certain limits. All authors caution against immoderation: compression of the uterus or stroking of the umbilical cord so as to force the last possible drop into the child. By such means the natural amount of blood in the infantile body is increased to an extent never before present and produces an artificial plethora which in its turn may give rise to undesirable conditions. Aside from slight hemorrhage from the vagina (Porak), bloody vomiting (Violet), hemorrhagic discharge from the intestine, and prolonged, marked icterus, such excessive measures may produce cerebral hemorrhage, as was proven by Illing at autopsy. As a criterion for the proper time to tie and sever the cord midwives and even some physicians wait for the disappearance of the navel pulse. Ahlfeld has pointed out, quite correctly, that with strong respiration this may not infrequently be felt for a long time. A better indication that the reserve blood has been transferred sufficiently is the relaxation of the veins of the cord, which occurs much earlier and no less distinctly.

Of the **diseases of the umbilicus** we are first particularly interested in *navel hemorrhage*. This condition, *appearing soon after ligature and division of the cord*, is principally due to the cessation of certain functions which physiologically are dependent upon the arrest of the circulation in the cord. In the active newly born infant the umbilical cord bleeds but little or not at all even without a ligature when respiration starts energetically and the child cries. Under these conditions life-threatening hemorrhages are exceedingly rare. In premature infants with deficient respiration, and in mature infants presenting asphyxia of the first degree the umbilical vessels remain engorged after birth and the arteries pulsate powerfully. Effective activity of the lungs is therefore of essential influence upon the amount and

circulation of the blood within the umbilical cord and in this sense acts as a limitation. With the first dilatation of the chest the blood of the venous system is drawn out of the body toward the thoracic cavity and the contents of the pulmonary artery are drawn into the lungs upon their simultaneous expansion. The necessary result is a decrease of the blood pressure in this portion of the circulation and therefore in the veins of the cord. At the same moment this change also occurs within the area of the descending aorta and its terminal branches—the arteries of the cord—the supporting force and the action of the right ventricle now being absent. If such an absolute cessation of the circulation of blood in the umbilical vessels is possible the active agent must be the coincident contraction of the walls of the vessels. According to Hoffmann, in extra-fetal life this begins placentalward and proceeds gradually toward the navel. The latter is soon reached after birth while the intraperitoneal portion of the navel vessels remains filled with blood for some time. In animals after hemorrhage had been arrested for some hours post partum Hoffmann was able to renew the bleeding and to maintain it for any length of time by the introduction of capillary glass tubes into the arteries of the navel. Deep respiration and energetic contraction of the vessels may all the more readily attain their effect in the newborn as the height of the blood pressure, according to the investigations of Hoffmann and v. Basch, is of less significance than that of the adult. When these factors are inadequate there is necessarily a danger of subsequent hemorrhage from the navel vessels. The predisposition of asphyxiated and premature children is therefore obvious. The congestion of the navel vessels prevents a sufficiently powerful contraction of the vascular musculature. That, however, a deficiency of the latter alone may appear is proven by the occurrence of secondary hemorrhage in mature, vigorously breathing and crying children. In addition to the proper measures to increase respiration there must be a careful ligation of the umbilical cord. Secondary hemorrhage, particularly when it endangers life, occurs less commonly immediately after ligation and division of the cord than later. This is readily explained in the case of asphyxiated or premature infants by the incompletely developed respiration, which soon becomes weaker. A gradual loosening of the ligature may then occur, particularly if it has been negligently applied. Wharton's jelly in time yields laterally to the ring-shaped pressure of the ligature. The inequality becomes the greater the richer the accumulation of mucous tissue. This readily explains the greater tendency to subsequent hemorrhage of umbilical cords rich in jelly. The navel bandage, however, which is applied wet simultaneously, dries under the influence of the body temperature and of the bed and may become loose. Therefore, with a lack of caution in applying the ligature or with very stiff, inadaptable linen a displacement of the knot is at least conceivable. A special tendency to this is brought about by the unequal band compress separated merely by a simple knot. Finally, a narrow navel bandage readily cuts into the substance of the umbilical cord, a broad one only when there is much friability of the tissue. Such immedi-

ate loosening is especially dangerous as they easily escape immediate observation. As a sure preventive it is usual to tie the cord provisionally and after a quarter or half an hour to re-tie it permanently, as well as to watch the condition closely. In another place I have stated that the double ligature which is advised in the Prussian Book for Midwives is of little service and tends to become loose. There is greater certainty if the cord is tied but once by a simple knot and loop. Instead of the linen bands  $\frac{1}{2}$  to 1 cm. in breadth which are especially recommended for midwives those of wool of the same breadth and with the same power of resistance are decidedly better on account of their greater elasticity. Finally, the time prescribed by the German law before the permanent tying is to occur is too short. It is better to advise attention to the child after the mother has been carefully looked after, i. e., after the delivery of the placenta is terminated. This will require at least one hour. If the nurse then gives the navel a superficial examination before she leaves the mother there will seldom be an uncontrollable secondary hemorrhage. Since the last half of the past century there have been quite a number of propositions for the positive control of the cord. Even surgical incision and tying of the vessels has been believed to be suitable for introduction into general practice. It would lead too far to state all of the methods that have been proposed. Only one will be mentioned which was set forth about 1870 by the French school (Budin, Tarnier) and at first was received with much enthusiasm: tying by *India rubber bands*. These maintain a constant and energetic compression and are not subject to subsequent loosening. Later investigations in the Clinics at Leipsic and Dresden met with good results. Nevertheless this method has not been generally introduced into practice, possibly because its application by one person without assistance offers too great difficulty and demands great experience and skill. Naturally, to combat an existing hemorrhage only a new tying of the navel cord centripetally from the first can be considered. When the blood loss is considerable, to prevent collapse a plentiful supply of heat, the administration of wine, brandy with water, and hypodermics of ether and camphor should be considered. [Camphor in ether is very painful.—EDITOR.]

A second critical period for vascular hemorrhage occurs during the process of healing of the navel, at the time *when the vessels are detached—when the cord separates*. It is then a question whether the closure has occurred so far centripetally at the height of the umbilical ring that it is able to offer sufficient resistance to the flow of blood. As already mentioned the peritoneal portion of the navel vessels remains open for some time after birth and is filled with blood. The tubes of the vessels are as a rule obliterated by agglutination and adhesion of the intima. A thrombosis, which however appears to be rather exceptional, may lead to a favorable conclusion if other phenomena are absent. Deviations occur primarily through the different forms of infection. In gangrene and putrefaction the stump of the umbilical cord swells, the vessels dilate and again become permeable. Septic



infection of the navel wound leads to inflammation of the vessels whereby they are changed into a tube with rigid walls and existing thromboses are destroyed. In the same category probably belong such sequels as Buhl's disease and Winkel's disease.

Syphilis is particularly serious since either the general condition of the child in itself predisposes to septic and pyemic affections and as a matter of fact is very often associated with them, or changes are brought about which have an unfavorable effect or prevent closure of the vessels. In the latter connection luetic vascular disease plays a much smaller rôle than the focal affections of the lungs (pneumonia alba) and of the liver (interstitial hepatitis, gummatous nodules). The only prophylaxis consists in the prevention of septic infection, therefore strictly aseptic or antiseptic measures. The syphilitic new-born must also be speedily subjected to specific treatment. An immediate result, before the sloughing of the navel, can scarcely be expected. In the treatment of slight hemorrhage cauterization with silver nitrate and a compression bandage (preferably of adhesive strips) come under consideration. In severe hemorrhage surgical ligature is recommended, especially if the necessary material is at hand. Dubois's method of tying the navel has been found no less successful. Two hare-lip needles are passed through the navel at right angles to each other and the second underneath the first. A silk thread is then twisted around each needle, partly in the form of the figure 8 and partly in a circle, and fastened at the base of the navel. When a severe blood loss has occurred the weakness and collapse must of course be treated in the manner previously indicated.

The vascular hemorrhages thus far mentioned are in the majority of cases limited; life-threatening bleeding is among the greatest rarities. A local hemorrhage can always be controlled by the treatment indicated. Accordingly various authors differentiate, under the term *omphalorrhagia*, a certain form of hemorrhage characterized on the one hand as parenchymatous from its diffuse distribution over the entire umbilical wound, and on the other as profuse, abundant, uncontrollable. We owe to Grandidier the first comprehensive description of this pathologic condition. According to this author the hemorrhage begins shortly after the cord separates, rarely earlier. The hemorrhage appears as if coming from a sponge. The lumina of the vessels, as the sources of the hemorrhage, cannot be seen even by sponging. Sometimes the hemorrhage threatens life from the onset; in other instances it is at first slight and increases by degrees, rapidly or slowly, continuously or intermittently. Local compression has but a transitory result at most. Grandidier calculates the mortality as over 80 per cent. According to the character of the hemorrhage death occurs soon after the onset, mostly after a few days or at the beginning of the second week of life; more rarely after a longer duration, in the third or fourth week. The principal accompanying symptoms are anemia, collapse, or gradual loss of strength. Grandidier regards such hemorrhages as idiopathic. This naturally can be stated only of those cases in which every other symptom of

disease is absent and the inability to control the hemorrhage is the only reason for assuming a hemorrhagic diathesis,<sup>1</sup> that is, a deficient coagulability of the blood. But these diffuse, uncontrollable hemorrhages are very rare, especially in hemophilic families. On the contrary, the majority of cases show other signs and symptoms indicative of a constitutional disease and accordingly hemorrhage from the navel must be regarded as secondary and symptomatic. Of special import in this connection are marked jaundice associated with some degree of cyanosis and early somnolence. Sometimes the symptoms on the part of the digestive tract are primary: simple dyspepsia, vomiting diarrhea. In the further course of the affection there are hemorrhages from other regions; stomach, intestines, cutaneous ecchymoses or large suggillations. Acute septicemia should first be considered. Most of the cases have occurred in the pre-antiseptic period and in foundling hospitals. With the improvement of the hygienic local conditions it has been possible to diminish the previously alarming frequency and in consequence of improved antiseptic treatment of the navel such affections have become exceedingly rare. The results of pathologico-anatomical and bacteriologic researches in the last decades have confirmed the view that these affections are of septic origin. The most serious effect of congenital syphilis is that it favors a possible septic infection. Whether, in addition, a substantive etiologic importance must be attached to this uncontrollable, diffuse hemorrhage has not yet been decided. This view, first propounded by Behrend, and later by Mraček and others, has been decidedly weakened in the last decade by the demonstration of streptococci. The chief element in the treatment of the affection consists in a careful observation of prophylaxis, therefore cleanly, aseptic care of the navel. The therapeutic measures to arrest hemorrhage are the same as have already been prescribed in the treatment of hemorrhage after the cord has separated: first compression, then acupuncture or encircling of the navel by means of two hare-lip needles. A solution of iron chlorid, formerly so often employed, on account of its strong corrosive action will probably be of use only in exceptional cases when no other aid is at hand. More advisable in recurrent hemorrhage from the stitched canal is the application of plaster of Paris. In addition to this local treatment injections of gelatin have recently been employed.

The *process of healing of the navel* corresponds clinically as well as pathologico-anatomically with the course of the healing process of anemic necrosis of the skin. The tissue of the umbilical cord is dependent for its nutrition entirely upon the circulating blood in the three vessels of the cord. A substantive supply is first produced fetalward from the boundary of the skin and amnion by an independent vascular net. From the superior and inferior epigastric arteries, from the amnionic sac with the urachus, and from the furrow of the liver along the vein of the umbilicus small vessels pass to the umbilical ring and by ramification and anastomoses they form in its immediate surroundings the subperitoneal arterio-umbilical circle from which

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<sup>1</sup> See Vol. II, "Diseases of Metabolism and of the Blood," p. 388, *et seq.*



branches pass through the umbilical ring and the linia alba to the surface and disappear within the subcutaneous and cutaneous tissues of the navel skin, forming capillaries. Superficially this vascular apparatus terminates precisely at the point of insertion of the amnion. Centrally, as a rule, the boundary zone has the form of a funnel-shaped depression, as the skin for a distance averaging  $1/2$  to 1 cm. continues upon the umbilical cord, that is, its tissues enter into the skin of the navel. This substantive vascular system is entirely uninfluenced by the exclusion of the placental circulation. With this change the only source of nutrition ceases at once, the tissue of the umbilical cord dies, and with sufficient air undergoes a gradual drying or mummification. The separation of the dead from the living tissue occurs through a demarcating inflammation. Epstein and Eröss have recently denied this fact. It is true that with an entirely aseptic course there is usually no marked reddening of the immediate surroundings. Nevertheless, aside from the fact that in the desquamation of necrotic foci this is usually poorly defined in the new-born in the first days of life it may even be concealed by the overfilling of the cutaneous vessels. Furthermore, the extensive emigration of white blood corpuscles has been frequently proven. Under their influence the amnion is first loosened from the skin of the navel, then the artery, and lastly the vein. In the normal and vigorous child this process is usually completed by the fifth or sixth day; in debilitated and premature infants somewhat later. In contrast to the original thickness of the umbilical cord only a small granulating wound remains which, through the shortening of the intraperitoneal umbilical vessel strands, simultaneously retracts at or below the level of the abdominal cover. After the umbilical cord has fallen off the invagination continues somewhat more rapidly. The progressive decrease in the wound is usually terminated by the twelfth or fifteenth day. The cicatrix is finally sunk in the umbilical groove, which is closed superficially by an upper and lower fold of skin.

As regards the nature and rapidity of the healing process of the umbilicus the funnel-shaped depression of the necrosed tissue in the integumentary portion of the umbilical cord is of great importance. The formation and position of the zone of demarcation resulting therefrom in relation to the superficial boundary of the amnionic skin form an essential means of stimulation of all of the active factors in the sense of a diminution of the wound, therefore in the shriveling desiccation of the cord and particularly in the retraction of the intraperitoneal navel vessel strands. Even with an outwardly normal length of navel skin this depression varies. On account of the smallness of the space deviations of  $1/2$  cm. play a great rôle. This explains why in the one case the entire healing of the wound appears to occur by depression and but little of the boundary remains for the later covering with skin, while in another case which may be externally quite similar the inverse occurs. The well-nourished fetal tissue may exceptionally reach to the superficial boundary of the amnion and even beyond. The latter condition is known as *resistant vascular stump*. Under these circumstances



the wound which remains after the cord separates is the larger; the further closure occurs almost exclusively by secondary skin formation from the marginal area and accordingly is decidedly slower.

The results are similar when the superficial amnionic boundary is unusually near to the surface of the abdomen or is situated upon it (amnionic navel). On the other hand, the inverse condition—a particularly long cutaneous process with an insufficient depression of the necrosed tissue of the navel cord—is without importance and only results in an abnormality of the healed cicatrix and umbilical groove.

As in every wound healing under similar conditions—therefore by secondary intention—prolongations of this kind show here also a *great tendency to immoderate granulation proliferation*. External irritation either of mechanical or infectious nature is present in the new-born in excess. In this manner the so-called *umbilical fungi*—bright red, broad-based, more or less pediculated granulomata—originate which maintain a profuse seropurulent secretion. As a rule they attain the size of a pea or small strawberry and, in consequence of the intraperitoneal shortening of the umbilical vessel strands, are usually so concealed in the umbilical groove that only the tip is visible between the cutaneous folds. In very neglected cases a length of several centimeters has been reported. Spontaneous healing becomes exceedingly difficult although by no means impossible. Küstner, Hüttenbrenner, Villar, and Pernice report instances of a delicate although faulty epithelial cover. Also, in adults, *mollusca pendula* originating in the depth of the umbilical groove find their simplest explanation in this manner (Heinecke, Küstner). Prophylactically these conditions are best met by the careful observation of cleanliness and early stimulation of the healing of the wound: touching lightly with a dilute silver nitrate solution, the use of astringent powders or salves (dermatol, iodoform, sanoform, etc.). Repeated cauterization of small proliferations with the silver nitrate pencil and separation of larger ones by the scissors after previous tying with a sterile silk thread bring about the most rapid results.

In addition to these tumor-like structures in the umbilical wound which are characterized microscopically as pure granulomata, other growths appear in the first weeks of life which macroscopically have the same form but the structure of which comprises epithelial tissue either in the form of cylindrical surface epithelium or tubular glands and smooth muscle fibers in large amounts. These in combination with adenoid interstitial tissue usually have a peripheral cover resembling the intestinal mucous membrane and corresponding with the interwoven bundles of the central zone. The first accurate investigations we owe to Kolaszek and Küstner. The latter author considered them substantive proliferations originating from the allantois passage or its residues which have remained permeable up to the navel ring, and called them *adenomata*. Kolaszek suspected the matrix in the tissue of the ductus omphalo-mesentericus and regarded the simple protuberance of the walls more likely as a persistent vitelline duct. This view was confirmed by the later

investigations of Schulze, Ohlfeld, Siegenbeek van Heukelom, Tillmanns, Pernice, etc. According to the extension of the malformation two varieties may be differentiated. Most commonly there is a retention and further development of a peripheral portion in the course of the navel ring and the zone of demarcation. Centrally the vitelline duct is entirely destroyed and the intestine completely closed. After the cord falls off there exists within the navel wound a canal with a blind termination proceeding outward toward the peritoneum. With the increase of intra-abdominal pressure due to beginning intestinal digestion, or perhaps more rapidly in consequence of crying, vomiting, pressure, etc., this depression is gradually forced outward and in time a protuberance occurs. The previously internal mucous membrane covering then becomes the external lining membrane and the muscular wall, formerly the peripheral part, becomes the central trunk. Contraction near the point of insertion, inflammatory hyperplasia of the upper segment in consequence of venous stasis, mechanical or infectious irritation, soon give to this protuberance a polyp-like appearance. The second form, which is much more rare, is due to the persistence of the omphalo-mesenteric duct with opening into the intestinal canal. The protuberance here appears more or less like the finger of a glove, cylindrical. At the height of the growth there is a small opening from which mucus and the contents of the intestine sometimes exude. Transverse section reveals first, from without inward, the intestinal mucous membrane, then smooth muscle, and lastly the mucous membrane. This development is in conformity with the microscopic structure and the designation proposed by Siegenbeek van Heukelom and Pernice as *intestinal ectopia or prolapse of the diverticulum* is well justified. Accurate knowledge is naturally furnished only by microscopic examination, but the macroscopic appearance reveals certain peculiarities in contrast to granulomata. This is particularly true in regard to the character of the secretion, which is not seropurulent but mucoid and thready. Also the development occurs gradually, as is apparent from the previous description, and is limited to a certain maximum. Finally, these intestinal ectopias never occur in the first days of life but as a rule about the third week.

Tillmanns and Weigert have expressed the opinion that similar formations may originate from the stomach and especially from the region of the pylorus. They base this view upon the careful observation of a case in which the secretion was of a distinctly acid reaction and the structure of the mucous membrane corresponded to that of the pylorus. Quite a number of hypotheses have been advanced as to the method of development. Siegenbeek van Heukelom has opposed this view and demonstrated that the structure of the mucous membrane which has been described is generally distributed in the intestinal canal in early life and not until the beginning of bile secretion do Lieberkühn's crypts and beaker cells appear. Accordingly the finding of Tillmanns and Weigert is constant in the intestinal ectopias which originate from a partial persistence of a peripheral portion of the vitelline duct and the other change is only noticeable with a complete retention.



Naturally, prophylactic measures cannot be employed against these structures. Therapeutically the best measure is excision after tying, the latter process being necessary on account of the medium-sized blood-vessels situated within the nuclear zone.

The most frequent and important disturbance of the process of healing of the umbilicus is *accidental wound infection*. A special predisposition for its development is assured by the principal factor of anemic superficial necrosis. With a certain amount of moisture—and in the new-born the frequent wetting of the diapers and of the navel bandage is sufficient—the necrosed tissue supplies a particularly fertile nutritive medium for the bacteria of decomposition. No less favorable are the conditions for the colonization of pathogenic microorganisms, streptococci, staphylococci, those of erysipelas, tetanus, etc., for within the range of the point of separation, on the border of the well-nourished and active tissue there is a zone of deficient nutrition and lessened resistance. On account of the active contact with the surroundings (strong vascular filling, cell proliferation, cell emigration) the bacterial products of metabolism are very energetically absorbed and new paths are opened to the aggressive activity of pathogenic germs by the establishment of numerous lymph spaces brought about by the separation of dead tissue.

The transmission of deleterious products occurs wholly by contact. Not only soiled hands, dirty scissors, bandage material, and the like, but contaminated bath-water, bath-tubs, and sponges are responsible. According to present views infection by means of the air is always secondary. In pre-antiseptic times septic disease of the mother played an etiologically great rôle. The most numerous and severe cases of navel infection were observed during epidemics of puerperal fever. Therefore it was not surprising that with slight knowledge of the cause of the disease an intrauterine transmission on the part of the mother was first considered and puerperal fever of the new-born was spoken of! Only with the founding and the further development of the theory of wound infection was the untenableness of this view apparent, and the more so the greater the results of careful prophylaxis. Children of healthy mothers were even more often and more severely affected, whereas, with puerperal fever in the mother, the process of healing of the navel in the infant was entirely without reaction. But the influence of a septic infection of the maternal genital organs cannot be altogether excluded. This influence, however, arises chiefly from the fact that the lochia abound in pathogenic organisms, the transmission of which to the navel of the new-born is greatly facilitated when the same nurse is employed for both mother and child. Furthermore the lochia of the normal lying-in woman from the third day are a veritable breeder of bacteria of decomposition. Finally, there is the possibility that the child may have introduced septic material into its own body from the septicly diseased organs of the mother. Theoretically this can certainly not be denied. Notwithstanding the fact that on account of the repeated cleansing of the infantile body a further retention appears to be scarcely



conceivable, if this should exceptionally be the case, in contrast with the frequent opportunity of contact in the first days of life this mode of transmission plays a very subordinate part.

A positive differentiation of the infectious diseases of the navel according to the nature of their origin is clinically impracticable. Apart from the unanimity of the pathologico-anatomical course there is almost always a mixed infection. Therefore the former division, according to the degree and extent of the local changes (*blennorrhoea, omphalitis, ulcer*, etc.), is of little use as the possibility of a general infection is not sufficiently considered. In practice the division into *locally limited infection transmitted to the rest of the body and general septic or pyemic disease of the navel* appears to be the best and simplest. Primarily the *putrefactive destruction* of the necrosed remains of the umbilical cord—the gangrene—remains localized. With the dissolution of the decomposed masses the danger to the organism of the intake of toxic substances is removed. The development of strong granulation forms a positive barrier to a later infection, which only occurs through injury. It is true the bacteria of decomposition furnish a favorable soil for the colonization of virulent pathogenic microorganisms, but even these septic germs and their diseases may be limited to the navel and its immediate vicinity. Why and when this happens is little understood. We usually assume that either the germs themselves possess an attenuated virulence or that the child is less susceptible and its tissues offer greater resistance. The latter is especially true of robust infants in contrast to the feeble, premature, or luetic. The external signs of contamination are most frequently decided redness and swelling of the immediate vicinity either greatly beyond the norm or continuing after the separation of the cord. In the former case the local changes may occasionally assume such a degree and extent that we are justified in speaking of a phlegmon of the navel and of the wall of the abdomen. Simultaneously there is usually a more or less increased secretion of pus with marked proliferative granulation, especially after dissolution of the cord. The position of the wound, in the depth of the navel groove, by adhesion of the superficial cutaneous folds readily gives opportunity for the retention of secretion. A wider distribution of the destruction to the skin of the navel, whether in the form of a purulent process (ulcer) or of gangrene, is much more rare. So greatly feared in the pre-antiseptic period, this condition now occurs only occasionally in debilitated and syphilitic children. All of these varieties may terminate in complete recovery with proper antiseptic treatment. On the other hand, with a prolongation and greater distribution of these local processes it is readily comprehensible that the danger of a general infection is increased. Nevertheless, the development of a peritonitis by continuity, regardless of the small layer of separation, is very infrequent. The septic virus in the navel of the new-born has a much easier means of direct communication with the interior of the body in the numerous lymph tracts which follow the navel vessels. These may also aid a rapid propagation to the interior without conspicuous superficial changes

in the navel wound and its surroundings, while the process of healing, on the contrary, apparently occurs without reaction.

Wharton's umbilical cord tissue continues immediately and uninterruptedly through the umbilical ring intraperitoneally in a broad and distinctly developed adventitia of the navel arteries. Successful injections have likewise revealed a direct connection of a rich, narrow-meshed net of lymph vessels. The conditions on the part of the vein are less favorable, a special adventitia being absent. The perivascular connective tissue terminates at the height of the umbilical ring. Here the fibers of the rectus fascia reach the true vascular wall (the media), without, however, joining or intimately associating, so that the vascular tube alone passes through the *linia alba*.

The lymph vessels in the perivascular layer, although in themselves somewhat broader, in comparison to the arteries along the vein are decidedly less numerous. Therefore anatomically the propagation of a septic virus corresponding to the course of the artery is decidedly facilitated—a fact which finds confirmation in pathologico-anatomical investigation, which shows that *periarteritis* and *arteritis* are most common occurrences in themselves as well as the causes of a general infection. *Periarteritis* may remain a simple cellular tissue inflammation, but may also lead to suppuration and to the formation of phlegmons or abscesses. The latter are situated below the navel, between the peritoneum and the abdominal fascia, extend usually only to the vertex of the bladder and open into the navel wound. A further distribution to the base of the bladder or to the scrotum and the inguinal region is very rare. The transmission to the vascular wall may occur outside of the navel ring, sometimes within or beyond, or occasionally at a distance of 1 to 2 cm., and with a decided extension in the perivascular connective tissue may not occur at all. *Arteritis*, like *periarteritis*, does not tend to distribute itself centripetally, without limitation; on the contrary, it generally terminates at the curve of the vascular strands in the region of the bladder. The further continuation to the retroperitoneal connective tissue upon the anterior side of the vertebral column, thence to the abdominal organs, and finally to the thoracic cavity is certainly exceptional. It is a remarkable fact that the development of *peritonitis* in connection with isolated disease of the arterial strands is comparatively subordinate. In the autopsy reports the lesions of lobar and lobular *pneumonia* with consecutive pleurisy decidedly preponderate. The causal connection readily becomes clear if the *vasa vasorum* are invoked in explanation. Not rarely these pulmonary affections were the only complication. All other organic lesions were more or less combined. As in the other general infectious diseases the organs principally affected were the liver, spleen and kidney. All of the stages from a beginning cloudy swelling to complete softening were observed. With extensive disease of the liver jaundice is correspondingly conspicuous. In the later stages of insidious chronic cases apostematoid foci, due to embolic propagation, may appear at various points. Arthritic suppuration and periostitis have been mentioned. Finally it must be stated that complicating diseases of the brain in the form



of diffuse encephalitis and meningitis and exceptionally isolated focal disease have been found.

Septic inflammation of the *vein of the umbilical cord*, as already mentioned, is much more rare. It may occur simultaneously with that of the arteries or alone. Here also the transmission to the vascular wall occurs in the vicinity of the navel wound or at a greater or less distance from it. Corresponding to the direction of the blood current the phlebitis endeavors to extend itself more to the central portion. From the point of transmission to the vascular wall the disease usually extends to the liver, and cases in which the vein has been found greatly altered in its entire length are by no means rare. Accordingly, in the further course the distribution by means of the circulation should seemingly be prominent. It is peculiar, however, that here the development of peritonitis is decidedly more common. More comprehensible is the early and marked change in the liver. According to Widerhofer this appears as a septic inflammation of Glisson's capsule or as a phlebitis of the portal vein. Hepatogenic jaundice, which is the chief indication of this condition, has depreciated as a differentio-diagnostic sign on account of its similar occurrence in primary arteritis and periarteritis. The other lesions coincide with those found in diseases of the artery and have already been mentioned.

Although the first propagation of the septic virus occurs entirely by way of the lymphatic tracts the further distribution rarely maintains the character of pure sepsis; on the contrary, by the implication of the blood channels it more often presents that of a mixed infection, of a septicopyemia.

The great predisposition to general infection which is furnished by infectious diseases of the umbilicus in the new-born is particularly evident in a review of the publications of the pre-antiseptic period. In the foundling asylums and maternities the mortality from septicemia and pyemia was enormous; during the time of puerperal epidemics almost devastating. Besides the rapidly progressive cases of so-called blood decomposition there were also phlegmonous, erysipelatous, and gangrenous inflammations of the surface of the body; multiple inflammations of the serous membranes, bones, and joints were very common, and even gangrene of the fingers, nose, lobes of the ears, and of entire extremities were not infrequent occurrences. Epstein, as lately as the middle of the seventh decade of the last century, reported the total mortality of the Foundling Asylum in Prague as 30 per cent., of which the majority gave a pathologico-anatomical diagnosis of septicopyemia. The betterment of these exceedingly deplorable conditions began with the minute investigation of the cause and nature of wound infection and increased as the observation of proper prophylactic measures gained prominence. Sporadic results were noted toward the end of the same decade. The real turning-point occurred about 1880, at the time of the general introduction of asepsis and antisepsis into obstetrics. With the decrease of puerperal fever the amount of the transmitted virulent material was lessened. The principal effect, however, was occasioned by a change in the general views,



which assigned the greatest importance to the danger of contact infection and accordingly regarded absolute cleanliness of instruments, bandages, the hands, etc., which came in contact with the wound as a primary necessity. Epstein reported in 1888 that, thanks to the observation of precautionary measures, the total mortality in the same Foundling Asylum in Prague had decreased to nearly 5 per cent., of which only about one-third were due to pyo-septicemia. Further advances have since been made and to-day severe cases of pyemia and septicemia in association with a local infection of the navel are exceedingly rare even in foundling asylums and maternities and are only observed in completely neglected and destitute children. While in a comparatively short time the danger of the more serious complications was decidedly decreased, much was still lacking for their complete disappearance. Eröss, in his publication from the first obstetrical clinic of Budapest in 1890, stated that among 1,000 infants only 320 experienced an uninterrupted healing of the navel; the remaining 680 cases showed mild or severe deviations and sometimes fatal disease; or, reckoning by the temperature curve, 43 per cent. showed an increase which, in 51 per cent., therefore in more than one-half, could be referred to infection of the navel. This still great morbidity was confirmed by a similar compilation from the second obstetrical clinic in Budapest (Tauffer). Results from other clinics (Innsbruck, Halle, etc.) are much more favorable although still remote from what can be regarded as ideal or even satisfactory. As the result of these investigations the question of infectious navel disease, its treatment and especially its prevention has recently been renewed. Many changes of the methods formerly in vogue have been proposed and tested in practice. Although a certain difference of opinion still exists as to individual methods all authors agree to the principle that continued improvement can only be attained by means of the strictest prophylaxis.

The recognition of infection from the local changes is easy after what has been stated. The physician need only examine the navel carefully from time to time. When transmission of phlegmonous inflammation to the arterial vessels occurs these, corresponding to their divergent course below the navel, may occasionally be felt as hard, sensitive strands. Hennig's observation—retraction and marked pallor of the triangular area of the abdominal skin situated medially, perhaps with a streaked redness of the lateral borders over the arterial strands—at the time regarded as characteristic, has not been confirmed by other authors. Periarterial suppuration produces swelling and redness of the overlying abdominal skin. In perforation into the umbilical wound a suppurative fistula develops, the contents of which may be discharged by pressure from the symphysis or spontaneously by an increased action of the abdominal muscles (crying, vomiting).

Of particular interest is the subject of an already developed general infection. We must always be prepared for this if the process of healing of the navel is not normal. As already mentioned, however, it may occur without any external evidence and when the wound apparently heals without reaction.

First, septicemia! Most important is the relation of the body temperature. As all other substantive causes of fever on the part of the intestine, the lungs, etc., in the first days of life, are essentially subordinate, every rise of temperature is suspicious, especially if it is continuous and has the character of continued fever with slight morning remissions. In how far the effect of absorption or intoxication can be considered depends upon the influence of local antiseptic measures. Simultaneously the pulse is decidedly increased. A possible disproportion to the height of the temperature in the new-born, however, does not have the same differential-diagnostic importance as in the adult. Very conspicuous is the early impairment of the general condition: at first restlessness, whimpering rather than crying, suffering expression, and finally somnolence, sopor, coma. With marked cerebral irritation or disease of the brain there is a typical muscular restlessness in the form of tremor of the arms or tonic and clonic contractions. In peritonitis the infant lies with the legs and body quiet but twitches upon any contact. The ingestion of food is more or less limited, either in quantity or by subsequent vomiting of that which has been swallowed. In addition, in the further course there is frequent and at times very profuse watery diarrhea (septic gastro-enteritis). Finally, especially in the severe forms, hemorrhages into the skin from the mucous membranes of the nose and mouth, hemorrhagic vomiting and bloody discharges may occur.

In this manifold, readily recognizable form septicemia of the new-born occurs only exceptionally. As a rule the individual sequences are much more limited in number as well as extent. Occasionally there is only the simple picture of a dyspepsia or enteritis: The temperature is not elevated; with a constant decrease in weight the child gradually becomes feebler or may perish in a few days in profound collapse. In cases of this kind the digestive disturbance present may readily be regarded as the principal factor; or the cause of death, particularly where debilitated or premature children are concerned, is registered under the common designations atrophy, marasmus, debilitas infantum. A similar error may occur with a febrile course when the symptoms of individual organs are particularly prominent. Here, besides the digestive tract, the respiratory organs must be considered, more rarely the brain and the cerebral membranes. Such errors can only be averted when the danger of a general septic infection during the time of the process of healing of the navel is considered and when it is remembered that a substantive disease of any of these organs during the first days of life is essentially rare.

The duration of the disease depends upon the severity of the primary infection; that is, the rapidity of the distribution. There are cases which terminate fatally within a few days and others in which the entire pathologic process is subject to gradual development, death being postponed until the second or third week.

*Pyemia* differs from septicemia by the appearance of metastatic suppuration due to thrombus displacement. Naturally, here, as in other conditions, the lung is primarily endangered. If the pneumonic focus is close to the

pleura the development of a purulent pleurisy, an empyema, is the immediate consequence. Smaller particles which readily pass the pulmonary circulation may occlude the capillaries of the muscles of the extremities. Next in order are the skin, epiphyses of the bones, joints, the internal organs, and finally the brain. Aside from the extension of the suppuration the prognosis depends principally upon the organ affected. If the vital organs are uninvolved and the strength of the child continues the process may terminate in recovery. Important in a clinical respect is the fact that the development of pyemia may occur quite late, even several weeks after birth, and may be observed a long time after the umbilicus has healed.

*Therapeutically* the principal factor is the careful observation of the necessary prophylaxis. Absolute cleanliness of the hands and of all articles, instruments, ligature material and bandages which come into contact with the navel is the chief requirement, not only when tying and severing the cord after birth but also in the later treatment, whenever the bandage is changed. These measures in the majority of instances are [too often left, Editor] under the control of the nurse. In those cases which are under the direction of the physician it is impossible for him to supervise the subsequent dressing of the navel, especially if the exclusion of moisture is to be regarded as an important factor in hindering putrefaction, as the bandage must be immediately changed when it has become wet. Nevertheless, every physician can do much to maintain the necessary precautionary measures. His attitude is an example for the nurse of what is to be done and left undone. Although the nurse may depart from the fundamental principles which have been given to her in her very brief course of instruction, the broadening of her later manner of thought is more greatly influenced by the actions of the physician than by the laws which control the situation. Professional interest increases the care, professional indifference leads to negligence. If the physician sees to it that the scissors and bandage material are thoroughly sterilized before use and placed in an antiseptic solution the nurse is less likely to neglect this precaution when away from his observation. More important even is his supervision of the nurse. The bandages are not only to be clean but are to be kept in a proper manner. The child should always be looked after before the mother. If, for certain reasons (urination, catheterization, movement of the bowels), this is impossible, the nurse must afterward thoroughly cleanse the hands. This precaution should also be observed when the nurse has handled the bed-clothes of the mother. A change of the navel bandage must never be made without a previous cleansing of the hands. Touching and manipulation of the navel cord or of the wound with the fingers is to be strictly avoided. If the physician gives this advice to a person who has not nursed for him previously, in the presence of the mother or one of the relatives she is more likely to feel herself under constant surveillance and will endeavor to prevent negligence. Every physician, however, even during the lying-in period, should carefully observe the condition of the navel. If now and then he informs himself of the condition of the navel wound, best before the child



is bathed, this serves as an important incentive for strict nursing and gives him an opportunity to test the care and skill of the nurse in applying the bandage.

The rules for the care of the navel have been essentially changed and improved in the last few years through the stimulation consequent upon the publication of Eröss from the Budapest Clinic. Previously it was generally held that the remaining stump of the cord should have a length of about four fingers, but for the more certain prevention of gangrene and to aid mummification a greater shortening has proved to be exceedingly advantageous. Two methods are of about equal merit. The one introduced by Ahlfeld consists of tying with a sterile naval ligature 1 cm. from the skin of the abdomen and the severance of the navel cord about  $1\frac{1}{2}$  cm. above it. Martin goes somewhat further than this by applying a strong silk thread close to the skin around the cord and separating the cord about 1 to  $1\frac{1}{2}$  cm. beyond. The cautery scissors employed by the latter author to prevent subsequent hemorrhage have been proved by later investigations (Stolze) to be unnecessary provided the tying is done with the proper care and with the necessary precautions. Both authors, Martin as well as Ahlfeld, accomplished this shortening by two different tyings. The cord was first tied in the manner already mentioned and after bathing a second cutting of the cord to the measure previously indicated was made. Both claim as the advantage of this interval that the cord contracts somewhat, the superficial layer dries, and a later loosening of the ligature is less to be feared. The increase of respiration due to the crying of the child in the meantime, as well as the greater progress of vascular contraction, is also of value. Upon the other hand this second operation has the disadvantage of doubling the danger of infection. The shortening to the required extent at once upon severing the cord certainly appears more advantageous. If the first tying is done provisionally by loops and in an hour is definitely fastened and two hours later, before leaving the patient, is examined again, the same purpose is attained. In my experience cases of subsequent hemorrhage have not occurred in recent years under this method.

In speaking of the equality of the two methods this was meant to indicate particularly the definite results and the absence of any complication, especially of disease of the navel. As to the course, Martin's process seems to be somewhat more favorable. Mummification of the stump of the cord and its demarcation take place more rapidly, separation occurs sooner, and finally an earlier and more rapidly progressive retraction of the skin about the navel is noticeable. With Ahlfeld's method a broad, raw, granulating surface remains after the navel falls off and depression occurs only gradually. Briefly, in Martin's process the duration of healing is one to two days less. On the other hand, there are a number of reasons which make its general introduction into practice less advisable. Ahlfeld showed that the silk ligature readily tears the tissue of the navel cord. Although, in comparison to the total number of cases, this was a very rare accident the possibility of such

a complication must nevertheless be considered. Its occurrence is especially unfortunate as a further tying around the cord cannot occur and the ligature of the navel skin necessitates a decided prolongation of the healing process and with this an increase in the danger of infection. It is better, therefore, to tie so far from the skin of the navel that if necessity arises another ligature may be applied, therefore from 1 to 1 1/2 cm. distant, as was proposed by Ahlfeld, and to employ a broad (0.3—0.5 cm.) cotton band. The fact that with Ahlfeld's method the healing process is one or two days longer is of no great importance as the wound, provided it has been kept clean, is then covered with granulations which serve as a powerful protective wall to further infection.

In addition to these methods of caring for the cord others have at times been attempted and advised (Peaudecerf, Kusmin, Keller, Dichenson, Porak, etc.). All modes of procedure have been proposed, from a careless division or squeezing without tying to the minutest closure by individual ligature of the navel vessels and a surgical knot of the cutaneous borders. While in the one the positive prophylaxis for eventual deleterious effects (hemorrhage) is absent, the others fail in other important requirements. All of them are more or less unsuitable for general practice and accordingly, after a short trial, have fallen into disuse. Their purely literary interest requires a further reference.

For the subsequent treatment of the navel the Prussian Text-Book for Midwives requires the wrapping of the stump of the cord in lint or cotton smeared with vaselin. The latter prevents too great adhesion of the cloth or of the cotton with the cord and a removal of the dressing without injury to the navel wound is possible. As Runge showed experimentally, about 1880, by the employment of fat or oil the drying of the remains of the cord is essentially retarded and a predisposition to the accumulation of germs therefore correspondingly increased. Furthermore, an accumulation of secretion readily occurs around the point of separation of the cord and hence aids a possible infection. This is not likely to be overcome by the addition of antiseptics as their disinfecting action suffers greatly by being embedded in fat and oil. For this reason the use of antiseptic fat ointments in wounds of similar nature which leave necrotic tissue after demarcation has been abandoned and in the care of the navel by this means a warning rather than advice is in place.

In many clinics the subsequent care of the navel occurs under the rules of pure asepsis, i. e., only sterile bandage material is employed, no antiseptics. This method is based upon the theory that, provided no unusual condition develops, the navel cord of the new-born at birth is free of germs, which was found to be correct by the bacteriologic investigations of Chalmogoroff. Theoretically considered the careful management by pure asepsis would be sufficient and thus the best results be attained; but this is only practicable in the hands of a physician whose soul is permeated by the spirit of asepsis and who never forgets that the slightest error is liable to render every advantage

illusory. The education of the nurse does not insure the required immunity. For this reason the treatment of the navel requires all the more consideration as the conditions for an accumulation of bacteria are much more favorable in consequence of the frequent contamination with urine than in wounds in other areas, and the change of the bandage which is so frequently necessary decidedly increases this danger. Finally, Chalmers, even with a sterile treatment of the navel and in spite of normal mummification, was able after a few days to demonstrate pathogenic bacteria. From this it is evident that in private practice and in institutions, whenever the care of the navel is entrusted to the nurse it is better to employ antiseptics. Most useful for this purpose are substances which combine a drying process with their disinfecting action. Both these properties are best fulfilled by alcohol. Ahlfeld uses alcohol after the bath and with every change of the bandage. I have followed his example for several years and am able positively to confirm his good results. The abdominal skin of the new-born bears alcohol well. The only precaution to be observed is that it does not come in contact with the external genitalia. Here, particularly in boys, it causes a decided edematous swelling. A permanent alcohol bandage, as is advised by Budberg, I do not consider necessary under normal circumstances. If, to aid mummification, the navel bandage is applied so that it admits the air the alcohol would evaporate very rapidly under the influence of the heat of the body. Therefore, to obtain a constant effect of the alcohol a more frequent change of the bandage or an air-tight covering is necessary, and both have proved to be unserviceable. In contrast to alcohol all of the watery solutions hinder drying on account of the addition of water. Besides, they do not enter so readily into all of the grooves and spaces. This is especially true of the pure disinfecting fluids, and therefore corrosive solutions, proposed by Schliep for the early period before demarcation is complete or before the navel has fallen off, even when greatly diluted, are objectionable according to our present views as they prolong the entire process unnecessarily. Among the antiseptic powders salicylic starch is most in use. At the beginning of the period of the antiseptic treatment of wounds the disinfecting action of salicylic acid was much favored, but on account of its slight solubility in the secretion of the wound it has deteriorated greatly in value. Still more unfortunate, as Rieck quite justly asserts, is the addition of starch, which by its tendency to crust formation readily retains the secretions in the space between the navel skin and the stump of the cord. The same is also true of clay, which has been advised as a dusting powder. The use of sugar might be of some value provided it could be obtained in a condition free from pathogenic germs, but actual disinfecting, bactericidal properties it does not possess. Of most service, according to many authors (Ahlfeld, Schrader, LeGendre, Varnier) are combinations of bismuth, especially of dermatol. In addition iodoform, sanofom, and itrol may be mentioned.

The question now arises: How often, or when, shall the navel bandage be changed? With the knowledge that the greatest danger of infection is



during demarcation, therefore before the navel falls off, and that it occurs by contact, the prophylactic appliance of an occlusive bandage during this period was considered for years. Tests were first made by Dohrn and later by Epstein, Eröss and others. Aside from the technical difficulty to attain an absolute closure on account of the frequent discharge of urine, the results were less than satisfactory. In agreement with the previous experimental investigations of Runge softening and purulent decomposition were much more observed. Although this decided the question of a permanent bandage the idea of limiting the change of the bandage has been maintained by many authors to recent times. Such physicians have the infant bathed only once, directly after birth, and the bandage is not renewed except when contaminated with urine. Although the omission is only a means to an end, other authors go still further and designate the bath before the cord has separated as harmful and caution against its use. Delayed mummification, more ready softening of the stump of the cord, and especially infection from the bath if the water used is not sterile or if the tub is employed for other purposes, for example, for washing the pads of the mother, are the reasons which are particularly set forth. Some authors, in confirmation of their opinion, emphasize the effect upon general development: Children who are not bathed in the first days are said to lose less in weight and to gain earlier. Investigations in regard to the effect of the bath were undertaken by Anthes-Schrader in Halle, and by Czerwenka in Graz. The results were entirely opposite. Children who were not bathed presented a proportionately much greater contingent of pathologic changes: softening, decomposition, febrile diseases, etc. As to a gain in weight no essential difference was determined in the Clinic in Halle, while Czerwenka was able to report decidedly in favor of bathed children. As it is accordingly proven that with proper care all deleterious effects of bathing can be positively prevented, such a statement is rather theoretic than founded upon practice. Anyone may readily convince himself by his own observation that actual moistening of the surface of the umbilical cord only occurs for one or two days. Notwithstanding this the mummification usually proceeds unhindered. Later the water runs off as from an oiled bottle, dropwise, a sign that with the drying there is associated fatty degeneration. The opportunity for the transmission of germs from the bath water is not serious and is only due to great neglect. In the investigations of Anthes-Schrader and Czerwenka the ordinary, non-sterilized water was employed. The principal danger lies in contamination by unclean vessels, which even among the poorest may readily be avoided.

The proposition of Epstein and others to add to the bath prophylactically a mild antiseptic (potassium permanganate, etc.) is superfluous after what has been stated. Actual value from this method is scarcely possible as the great dilution of these already weakened substances necessitates a very long time to develop their effects. Furthermore the danger must not be disregarded that the nurse, having confidence in the disinfection, may neglect the necessary observation of strict cleanliness.

Finally, an advantage of omitting the daily bath would be a less frequent change of the bandage. But even this is over-balanced by a series of disadvantages. That bathed children flourish better has already been mentioned. Furthermore, in the bath the wound is cleansed of the adherent secretion without contact, almost of itself. In contrast, the change of the bandage after contamination with urine acts as a trauma. Although Keilmann maintains the opposite opinion probably no practitioner will agree with him. Then the wholesome effect of the bath upon the skin! Frequent and vigorous <sup>1</sup> washing leads to intertrigo and hence produces new and no less serious ports of entrance for pathogenic microorganisms. Erysipelas, furunculosis, suppuration of lymph glands, phlegmons and, in girls, cystitis and pyelonephritis—pyometra, salpingitis—(Epstein) have been noted. By the desiccation of the cutaneous secretion, especially of the firmly adherent vernix caseosa, more or less circumscribed eczemas arise. In addition is the impression upon the laity and nurses, whose conception of cleanliness is embodied in the daily bath. To them the possibility of harm from this source is absolutely incomprehensible. Its omission will not be regarded as a stimulus for more caution and care in regard to cleanliness; on the contrary, it will soon cause negligence.

Therefore, with the necessary care the daily bath has nothing against it and much in its favor. In addition to the required change of the bandage after the bath it must be renewed whenever contaminated with urine. The long continuance of moist heat at the temperature of the body favors softening and decomposition all the more when absorption and evaporation are prevented by a rubber cover. The purpose of the latter, to protect the crib, is fulfilled without any harmful effects if it has an opening in front, which is also a reminder to the nurse to substitute dry diapers for the wet ones.

Particular care of the *umbilical cord* is necessary in the *birth of twins*, especially if the wound of one infant is infected. The most obvious precautions, care of the other child by another nurse and the employment of a separate bath-tub, can only be strictly observed in hospitals. In practice generally our only recourse is to attend to the healthy child always *before* the sick. In the interest of the former it is especially advisable not to bathe the latter, particularly if the illness is serious, although this hinders the transmissibility but little. In such cases regular washings with alcohol have always proved an excellent prophylactic.

For the local treatment of the wound antiseptic measures are in place. With extensive superficial suppuration dry powder dressings with a mildly absorbent gauze are employed (boric acid, iodoform, dermatol), and with distributed inflammatory infiltration moist compresses with potassium permanganate or acetate of aluminum. In cases of this kind an alcohol dressing in the form advised by Salzwedel has proved with me to be excellent and may be substituted by the dry dermatol applications when the symptoms

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<sup>1</sup> The normal desquamation of the skin and mucous membranes of the newly born forbids "vigor" in the treatment of the surface and the mouth.—EDITOR.



begin to improve. Subcutaneous abscesses between the navel and the symphysis, and later in other areas, require early incision. Other reliable measures to prevent the further extension and development of general infection are not at hand. We are limited to a proper nutrition and perhaps analeptics to prevent a threatening disability. This applies also to those cases which already show the signs of a general infection. A trial of Credé's salve is certainly not contraindicated, (?—Ed.) but to the present time I have seen no brilliant results from its use. Antistreptococcic serum therapy is not yet so far advanced as to justify confidence. Its trial, however, should not be neglected.

In addition to septic general infection the navel wound of the new-born plays a very important rôle as a port of entrance for the bacilli of tetanus. The disease which results from this—*trismus* or *tetanus neonatorum*—has already been described in the chapter upon tetanus, and therefore, to avoid unnecessary repetition, I must refer to that article.<sup>1</sup>

Only with cicatrization of the cutaneous wound is the external closure of the navel completed. That of the actual navel ring, through which the vessels pass within the linea alba, takes place gradually toward the end of the first year. The *tendency to hernia* which results from this during the early period does not affect the cutaneous cicatrix, but, on the contrary, occurs above, between the outer circumference of the navel ring and the stump of the vessels. Formerly the fundamental cause of this peculiarity was referred to the energetic retraction of the vessels of the cord which are intimately combined with the cicatrix, particularly the arteries. According to Robin, in the course of time there is even a mutual displacement of the individual layers of the vascular walls, especially of the media toward the adventitia, which brings about the transformation into solid fibrous strands which are actually found a few weeks after birth in the vicinity of the navel. Herzog has demonstrated by exact anatomic and microscopic investigation that the entire process depends upon a transformation of the fetal cellular tissue into rigid connective tissue, to which is added an obliteration of the navel vessels and a degeneration of their walls into tense fibrous tissue with destruction of the muscular elements. The incompleteness of the closure is a direct consequence of the original anatomic arrangement, and is later increased by *connective tissue contraction*. As already mentioned the arterial strands possess a distinct, well-formed, broad adventitia which is in intimate connection with the tissues of the linea alba and of the navel ring, while the vein is only loosely layered in the upper portion of the latter without a substantive adventitia. Although, accordingly, the transformation into rigid, coarse-fibered connective tissue causes the closure of the lower portion of the navel ring to be essentially firmer and more complete it must be added that the changes of the arterial strands begin at the navel and proceed toward the bladder. In the vein, the opposite direction is taken, from the liver to the navel. Within the abdominal walls the venous vessel is immediately adjacent to the arterial strands. The stronger and earlier contraction of the

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<sup>1</sup> See volume on "Infectious Diseases."



latter, therefore, must produce a corresponding dislocation of the former and approximate it to the lower circumference of the navel ring and remove it from the upper. In this manner there develops between the upper circumference of the navel ring and the remains of the vein a point of least resistance. This space, known as the umbilical canal, which is filled with loose cellular tissue, is closed peritoneally only by a narrow layer of connective tissue and externally by the skin. Great exertion of the abdominal press (screaming, vomiting, pressing, coughing, etc.) may readily protrude the intestines and lead to hernia, preventing a definite closure. Prophylactically, in the first three months it is always advisable to provide a certain amount of support for the navel by the employment of a bandage with an underlayer of cotton. For the treatment of navel hernia the application of an adhesive plaster bandage over a firm plate was formerly the preferred method; now injections of paraffin will probably be used.

### OTHER SOURCES OF SEPTIC GENERAL INFECTION

In the foregoing it has been shown that infection of the umbilical wound is the most common source of *septic general infection* of the new-born. In addition, there are other *ports of entrance*. Any **external injury** to the child may be subject to a similar fate and course. For this reason in the pre-antiseptic period pressure marks, etc., were greatly feared. Naturally, contamination may occur as readily post partum as intra partum since the same instrument, the hand, may produce the injury and infection. In the subsequent days of life these tissue spaces associated with intertrigo become serious. Their various mild and severe sequels have already been mentioned. The cutaneous changes of congenital syphilis also belong in part to this category. Finally, as in the adult, streptococci may permeate the exits of the glandular ducts, lead to a local septic inflammation, and be transmitted thence to the rest of the body.

A second path has its starting-point in various portions of the **digestive tract**. The oral cavity and the anus are chiefly predisposed since they are most frequently subject to direct contact. The careless introduction of the thermometer into the rectum, or of the nozzle for an enema, may be the immediate cause, and in the oral cavity unskillful washing is the most common. Apart from the delicate composition of the oral mucous membrane the marked desquamation which occurs within the first few days of life favors the development of superficial injuries. Through the limitation which cleansing of the mouth at birth has undergone in the last decades the influence of this source of infection has been greatly decreased. In relation to other portions of the digestive system the composition of the food must be considered. P. Müller was the first to voice the suspicion that puerperal infection of the mother may be transmitted to the child through the milk. At first bacteriologic investigation seemed to confirm this view. In the

puerperal lying-in woman the milk contains staphylococci (Escherich, Karinski); normally it is always sterile (Meissner). Bumm demonstrated cases in which the milk of the septic lying-in woman, notwithstanding repeated and minute investigation, was found free of germs, and Cohn and Neumann were able to cultivate bacteria and staphylococci from the milk of a healthy mother which probably had permeated from without into the gland. There is a possibility that germs may be transmitted to the child with the milk; nevertheless it has been practically proven that the milk of the septic mother may be taken by the child without injury (Runge, Wolff, Schütz, Biedert, etc.). A healthy digestive apparatus is therefore uninfluenced and only reacts to the smaller number of bacteria which come into consideration when diseased.

Septic microorganisms may also gain access by way of the **respiratory tract**. By aspiration of decomposed amniotic liquor or of the maternal genital secretion intra partum in premature respiration septic pneumonia may develop from which the child perishes soon after birth. This occurrence, however, is not frequent. Most children perish intra partum in consequence of severe complications; others born alive show only transitory catarrhal changes or remain perfectly well. For the differential diagnosis it is important to know that this is also the most frequent localization in the metastatic inflammation of umbilical sepsis. Here, apart from the corresponding symptoms in the mother the early development, even before the loosening of the umbilical cord, is decisive. Another means of transmission by way of the respiratory tract is by inhalation. Experimental investigations are but little conclusive on account of the overloading of the respired air with septic germs and the manner in which they were introduced in the experiment. It must also be remembered that the infective products had previously found sufficient points of deposit on account of the predominantly nasal respiration of the infant. From here, it is true, the process may gradually distribute itself further and deeper. Highly interesting and conclusive in this connection are the reports of an epidemic of septic pulmonary disease in the new-born reported from the Heidelberg Obstetrical Clinic by Gärtner, which he traced bacteriologically to infection from damp straw bedding. Such a process of distribution, however, is the rarest and usually beyond our intimate recognition.

Among the older authors the **transmission by the placenta** is assigned a great rôle. Although, in comparison to former times, this possibility has lost greatly in importance on account of the further development of the law of wound infection and especially of septic general disease, it cannot yet be altogether rejected. Among the cases which have been published only an observation of Orth's will stand the most exacting criticism in every detail. The necessary prerequisite is a corresponding disease of the mother during the puerperal period or at the latest intra partum. The fetus is still-born or perishes soon after birth. With the positive exclusion of all other sources of infection, especially on the part of the navel, the autopsy must reveal the

evidences of sepsis. The rarity of septic disease of the mother during pregnancy or parturition is due to prophylaxis. [The researches of Warthin, of Ann Arbor, on the transmission of tuberculosis through the placenta were not known to the author.—Editor.]

## DISEASES OF THE EYES

### GONORRHEAL INFLAMMATION

The *eyes*, like the navel, require careful attention after birth. The intimate contact of the palpebral fissure and its immediate surroundings with the maternal genital walls and with the adherent secretion, after the rupture of the fetal membranes, is the means of an infectious transmission which is all the more possible the slower the birth of the head. All pathogenic microorganisms in the maternal genital apparatus come under consideration, no matter under what circumstances they may be present. Most common and most serious is Neisser's *gonococcus*. Exceptionally the invasion occurs so early that the infant is born with inflamed eyes. Kruckenberg, Feiss, and others have reported cases in which the specific character was confirmed by the demonstration of gonococci. No less often the disease is observed within the first twelve to twenty-four hours. The intrauterine onset is here less certain, for according to experimental inoculation tests (Bumm) and in the individual phases in which infection occurs in adults there is a possibility of an incubation within a few hours. In by far the majority of cases the first signs are noted from the third to the fifth day. The explanation of this apparent delay is that the infectious secretion at first merely adheres to the external surface of the lids, the eyelashes and the borders, the internal and external canthus, and only enters the conjunctival sac later, when the eyes are opened, or by unskillful and particularly wet cleansing. According to the maximum period of incubation of gonorrhea a later affection can only be assumed by another form of transmission (fingers of the mother or nurse, unclean bedding, bed-clothes, sponges, bath water, etc.) whereby the germ-containing material may be transmitted from the mother, as well as from another child with diseased eyes or from any individual suffering from gonorrhea. Clinically the consequences are first revealed by an ordinary catarrhal inflammation. The palpebral space is slightly agglutinated. When it is opened a profuse, thin, turbid secretion containing numerous flocculi is discharged, often with a certain amount of force, in a stream. The conjunctiva is greatly reddened and swollen, especially in the region of the conjunctival fold. In the course of hours these inflammatory changes are rapidly increased. The eyelids, especially the upper, are reddened externally, with edematous swelling. The eye is always closed. The rigidity of the lids makes the opening of the palpebral space difficult. From there a thick, mucopurulent, whitish-yellow secretion oozes, the removal of which reveals the deep red conjunctiva with velvety swelling



and marked infiltration, and upon widening of the fissure new rolls constantly protrude. At first nothing can be seen of the bulbus. An accurate view is only possible by completely inverting the eye-lids. The marked changes are localized to the conjunctival fold. The conjunctiva bulbi is also involved and forms a wall-like prominence around the cornea. With the transformation of the secretion to a purely purulent character there is in a few days a gradual decrease of the rigid swelling. The lids become softer, the conjunctiva looser. In the region of the conjunctival fold the tendency to wrinkles is noticeable and in the tarsal portion papillary proliferation is particularly apparent. The secretion, previously abundant, decreases, gradually assumes a mucous character, and finally ceases. The fold-like and papillary proliferations may disappear without causing cicatrization. The duration of the disease varies between two and eight weeks. With a prolongation of the final purulent stage, however, it may assume a chronic character and recovery may be retarded for months.

**Pathology.**—Pathologico-anatomically gonorrheal conjunctivitis is not a surface affection. Here as in the genital apparatus the gonococci actively invade the epithelial layer by way of the cement substance to the subepithelial connective tissue. Bumm was the first accurately to investigate the individual phases of this process, which he has published in an explicit article. Upon the second day the epithelial layer may be completely permeated with diplococci. The immediate consequence is a correspondingly rapid and great inflammatory reaction of the vascular connective tissue (hyperemia, serous and cellular exudation). Vast swarms of white blood corpuscles collect in the superficial layers and permeate the epithelial cover to the surface. By this means the epithelial layer, which has been previously loosened by the invasion of bacteria, becomes still more relaxed or raised. At the limbus corneæ and on the border of the lids this process is sharply demarcated. The transitional epithelium remains uninfluenced by the action of the bacteria and its appearance is unchanged. After breaking through and loosening the epithelial layer the gonococci distribute themselves in long or short chains or round colonies and also to a certain extent within the superficial connective tissue layer. A deeper permeation is rarely observed and in form and course greatly resembles pre-formed lymph spaces. Under the influence of these microorganisms the inflammatory symptoms are greatly increased. The round-cell infiltration may finally extend 2 mm. below the surface and is so dense that cell approximates cell. The blennorrhœa has passed into the purulent stage.

Aside from the decrease of gonococci and the cessation of the irritative symptoms in the connective tissue of the mucous membrane the commencement of healing is characterized by the beginning regeneration of the surface epithelium. This covering, at first consisting merely of a simple layer of cubic or flat cells, by the rapid proliferation of the elements soon reaches a thickness of two or three layers, the uppermost being subject to a gradual deposit of epidermis. During the new epithelial formation the emigration

of the white blood corpuscles continues without any disturbance of the regular arrangement. With its completion the papillary body is entirely free of gonococci, which may only be found superficially between the individual epithelial elements. Renewed infection and accidents may interrupt and prolong the uniform progress of healing. In the third week of the disease the epithelium is usually intact. The entire conjunctival sac is finely clothed by layered pavement epithelium which is substituted by cylindrical epithelium after complete disappearance of the swelling and proliferation of the papillary bodies.

The principal danger of this affection, which is usually designated *ophthalmoblennorrhoea*, *gonorrheal conjunctivitis*, or *blennorrhoea neonatorum*, depends upon the transmission of the inflammation to the cornea. Superficial desquamation of epithelium in the region of the palpebral fissure, especially near the border of the cornea or below its middle, is by no means rare. So long as the process remains thus limited a subsequent damage is excluded. The early superficial turbidity is only transitory and finally disappears completely. Through these epithelial defects, however, the way is open for the generators of inflammation to the interior of the eye. Infiltration of tissue occurs and the necrotic destruction causes ulceration. If this remains within moderate bounds and soon yields to cleansing and healing the resulting cicatrization causes a corresponding turbidity of the cornea (leukoma), which decreases in intensity and extent with advancing age, but nevertheless, from its position in the pupil and its circumference, may cause a more or less decided impairment of sight. If the necrotic process continues, perforation of the anterior chamber of the eye is inevitable and is followed by further injury: prolapse and adhesion of the iris (leukoma adhærens, partial or complete staphyloma), anterior capsular cataract, protrusion of the lens and a part of the vitreous body with succeeding atrophy of the bulbus; or, by a distribution of the process to the interior of the eye a panophthalmia occurs. Thus there may be any stage of impairment of vision to complete blindness.

From the conjunctival sac the gonorrheal catarrh may pass to the nasal mucous membrane and from there distribute itself to the internal ear (gonorrheal otitis media). Diseases of the oral cavity, occasionally observed simultaneously with gonorrhea of the eye, may accordingly be referred to a substantive transmission, either during birth or, more readily, later by the hands of the nurse or of the mother, by mouth swabs, etc. Nevertheless, the possibility cannot be excluded that a germ-containing secretion from the eye may flow over the face into the mouth.

Constitutional affections characterized by the appearance of remote metastatic inflammatory foci in connection with a primary gonorrheal conjunctivitis are comparatively rare in the new-born. Here also the large joints, especially the knee, are most often affected. The acute onset (sudden, marked, sensitive swelling with fever) later passes into a prolonged chronic course. The tendency to suppuration and ankylosis appears to be decidedly greater than in a corresponding condition in adults. Similarly, periarticular

arthritic inflammation in the cavity of the joint, without exudation, and tendovaginitis may lead to motor disturbance. Finally, endocarditis must be mentioned—the severest but also the rarest complication—which usually occurs in association with gonorrheal rheumatism and only exceptionally alone, the clinical symptoms corresponding in the main to those of the adult form. [The small joints, mainly of the hands, also the cervical lymph bodies connecting with the nares and the mouth with consecutive general pyo-septicemia, may be met with.—Editor.]

**Diagnosis.**—The diagnosis of gonorrheal ophthalmoblennorrhoea depends upon the bacteriologic examination of the secretion, for which a simple smear preparation is sufficient. In addition to the characteristic diplococcus form, described by Neisser as bun-shaped, the arrangement into small and large colonies within the cell protoplasm is important. The number of gonococci present varies greatly in different cases and is not actually proportionate to the degree of the local inflammatory condition. In severe blennorrhoea they are often scant, in the milder forms numerous. The observation is also of importance that the bacteriologic investigation may occasionally be positive after a decided decrease of the final catarrhal symptoms; therefore transmission even then cannot be altogether excluded.

**Prognosis.**—The prognosis depends in general upon the complications. Although doubtful without these it is essentially improved by the early institution of proper treatment. Nevertheless, it is well not to be too sanguine, for notwithstanding all precaution and proper care eventual blindness has been observed. Essentially important are the general nutrition and the development of the child: In debilitated, premature infants and those enfeebled by digestive disturbances an affection of the cornea cannot always be prevented.

**Treatment.**—For *prophylaxis* we must differentiate between transmission at birth and that occurring subsequently. The former may be counteracted during pregnancy by antiseptic measures. Tamponage of the vagina with gauze saturated in a 1 to 1,000 solution of corrosive sublimate twice every week for five or ten minutes has proven in my hands more advantageous than injections. By this means we best succeed in penetrating the numerous folds and creases. Silver nitrate solutions even when greatly diluted produce marked irritative phenomena and are painful during pregnancy. More suitable are the organic silver salts, such as protargol, etc., which have a weaker action. Directly after birth the best prophylactic, proposed by Credé in 1881, is the dropping of a solution of silver into the conjunctival sac, which to-day is recognized by all authors as the only positive and reliable method. The statistics of some of the obstetrical institutions are particularly convincing. Previously from 9 to 15 per cent., and in the last decades of the last century sometimes as high as 50 per cent. (Haase), the morbidity after the introduction of Credé's method fell as low as 0.8 per cent. Unfortunately the early optimism that ophthalmoblennorrhoea could thus in time be completely rooted out proved to be premature according to



the later compilations from blind asylums. According to Cohn's report, within fifteen years (to 1895) there was a decrease in the blindness due to blennorrhea to 19 per cent., from 30 per cent. in former periods. In 1902 a similar report was made by Hirsch, who found that 12 per cent. of the cases of blindness in adults and 18 per cent. in adolescents were due to blennorrhea. These comparisons only consider total blindness. For a complete summary the cases of unilateral blindness and partial impairment of vision should be added, which would considerably diminish the difference. The comparatively slight success of the method in general practice is readily explained by the fact that 95 per cent. of all births (in Germany) occur not only outside of institutions but even without a physician. It is true that in the Prussian directions to midwives since 1892 Credé's method is demanded for women who "suffer from a contagious vaginal discharge." But in addition to the fact that the midwife is not capable of deciding this question there are also gonorrheal infections, especially of the mucous membrane of the cervix, in which the secretion from the vagina, which is always present in pregnancy, is not materially increased. The modification of the directions to read "with a purulent discharge," as is contained in the Bavarian law, is but little better. A change in general practice can only occur by relieving the midwife of responsibility and the obligatory introduction of Credé's method. The objection raised by some authors on account of subsequent consequences in non-infected children (silver catarrh) is overcome of itself, for even these authors admit that the development of such a condition may be due to an unskillful application of the method; therefore, with sufficient instruction this can be excluded. The obligatory introduction might at least be made dependent upon the permission of the father, whose refusal would relieve the nurse of responsibility [and the baby of blindness?—Editor]. So long as there are no definite rules physicians may do much to improve the condition. Here also, as has already repeatedly been emphasized, the fundamental principle is that the professional interest and behavior is a guide to the nurse for her commissions and omissions. If the physician, in a labor occurring under his direction, attaches value to the employment of Credé's method, the nurse when away from his supervision will be less likely to neglect it than in the inverse case, when she will suppose it to be superfluous. A favorable opportunity thus arises to teach the nurse the method or to show her the correct procedure. The custom of some practitioners to make the treatment dependent upon the positive report of the parents is not reliable, as untruthful statements are by no means unusual from a false sense of shame or ignorance of the serious consequences.

The method advised by Credé consists of the instillation of a drop of a 2 per cent. solution of silver nitrate. For accuracy his directions will be given verbatim: "After the cord has been tied, the child bathed, and the eyes wiped with a clean cloth and sterile water—not bath-water—until all cutaneous mucus adherent to the lids has been removed, and before the child is dressed, an instillation should be made. By means of two fingers each

eye is partially opened and a small drop of a 2 per cent. solution of silver nitrate, dependent from a glass rod, is brought in contact with the cornea and let fall upon the middle of the structure. All further treatment of the eye ceases. If in the next twenty-four or thirty-six hours a slight swelling or redness of the lids with excretion of mucus should result the instillation is not to be repeated. The glass rod should be 3 mm. thick, with rounded ends and smooth. The silver nitrate solution must of course be kept in a dark bottle with a glass stopper. The supply should be as small as possible (about 15.0)."<sup>1</sup>

It is important that the first superficial cleansing should not be done with saturated cotton, but with sterile cotton wrung from boiled water; otherwise the secretion, laden with microorganisms, may easily be flooded into the conjunctival sac. Instead of the glass rod a pipette is now in general use. The danger that several drops may be instilled and cauterize too severely may be avoided with practise. The point of instillation—the middle of the cornea, corresponding to the middle of the conjunctival sac and near the point of first involvement in the palpebral space—also appears to be important. The frequency of marked irritative phenomena, the so-called *silver catarrh*, which was observed by Cramer contrary to all other authors, may probably be explained by his special and more irritating technic. Cramer dropped the silver solution into the internal angle of the eye, then opened the lids, directed the solution over the eye by a lateral inclination of the head, and distributed it in the conjunctival sac by closing and opening the lids. His caution against Credé's method has in this way been explained and rejected by Leopold. The same antiseptic effect that is produced with a 2 per cent. silver nitrate solution occurs with a 1 per cent. solution, but with slighter secondary irritative phenomena. Runge, Gusserow, Fehling, Hofmeier, Leopold, and others have tested its employment extensively and obtained excellent results. After a practical experience of about ten years I can confirm this opinion and would advise the modification of Credé's method in practice. Watery solutions of silver nitrate become more concentrated after long standing by the gradual evaporation of the water and cause greater cauterization and more marked irritation. In city practice this disadvantage is readily overcome if the solution is always freshly prepared. The fulfillment of this precautionary measure in obstetrical practice in the country is more difficult and its observation by midwives can scarcely be expected. Zweifel therefore substituted silver acetate, which only dissolves to 1 per cent. at the temperature of the room, and with further evaporation throws down the precipitate. To prevent a marked corrosive action subsequent washing with a normal salt solution was tried. The prophylactic

<sup>1</sup> History repeats itself, also the neglect of its teachings. Exactly fifty years before Credé, Eisenmann—the same who was left in a dungeon twelve years by one of the trembling and rancorous German rulers—the founder of what is best known as Virchow and Hirsch's annual reports, recommended besides absolute cleanliness the use of a mild chlorine water. Nobody followed his advice, the babies got blind as before, and the blind asylums "profited."—EDITOR.

result has also been recognized by other authors (Tauffer, Hofmeier, etc.), but the absence of irritation has been contradicted. The same is true of the 20 per cent. solution of protargol (Zweifel) recommended by Engelmann and later by Piotrowski and Veverka, and weaker concentrations have proved unreliable. In addition to these reagents quite a number of others have been tested—salicylic acid, carbolic acid, chlorin water, potassium permanganate, iodoform,  $\beta$ -naphthol, resorcin, thymol, iodine trichlorid, zinc sulphocarbonate, corrosive sublimate, mercury citrate, ethylenediamin—but upon subsequent investigation have been found to be less serviceable in prophylaxis. Therefore, the experience of syphilographers and ophthalmologists regarding the specific action of the salts of silver upon gonococci seems to be confirmed.

For the prophylaxis of transmission in the days subsequent to birth the same rules are operative as have already been explicitly detailed for the prevention of navel infection; therefore scrupulous cleanliness on the part of the midwife, nurse, and mother, which applies to the hands as well as all articles which come into close contact with the eye! Beware of sponges; fresh absorbent cotton should always be used! Strict observation in regard to cleanliness of the bath-tub and particularly that it is not employed for washing the pads of the mother! Care of the child always before attention to the mother! The mother should have the child in bed only for nursing, and invariably before she takes the child her hands should be thoroughly washed. Finally, when gonorrhea is present the mother and the nurse should be expressly instructed in regard to the danger of transmission to the child as well as to themselves and to those about them.

If one eye is already infected the first requirement is to prevent the other from a secondary infection. Besides the observance of general rules the head of the child should be placed upon the diseased side, the hands tied, the normal eye cleansed always before the other, and the diseased eye wiped from within outward, which is most positively accomplished by the application of an occlusive bandage: cotton, gauze pad fastened with collodion or cotton, gutta percha tissue fastened by means of chloroform. This bandage must be removed twice daily to note whether the eye has become infected and is then renewed immediately. Some authors also advise the periodic instillation of 1 per cent. silver nitrate solution.

The beginning of local treatment of the diseased eye had best be expectant and limited to the avoidance of any great accumulation of secretion by its frequent removal and by mild disinfection of the conjunctival sac. After cleansing the external surface and with the palpebral space somewhat open the lids are everted and the conjunctiva cleansed by means of cotton or a gauze compress. The fluids employed for disinfection are the milder agents—boric acid, 2 to 3 per cent., hydrochinon, resorcin, etc.—also dilute solutions of stronger disinfectants—corrosive sublimate 1 to 5000, mercury oxycyanid 1 to 500, iodine trichlorid 1 to 4000, potassium permanganate 1 to 3000, silver nitrate 1 to 1000. This process of eversion



appears less irritating to the conjunctiva and particularly to the cornea than the method of distributing the fluid over the eye by friction of the eyelid. Careful cleansing by this means should be done at least twice daily. In the interval, every thirty minutes or an hour, the eye is washed externally with slight separation of the lids. In this stage the application of cold is advantageous in the form of cotton or gauze compresses moistened with one of the above disinfectants and cooled upon ice, and changed every three to five minutes. All bandage material should be burned after use. When the secretion has assumed a purulent character and the swelling of the lids has decreased to the extent that the eyes open voluntarily, usually from the third to the fifth day, a caustic astringent treatment may be begun. In severe cases the silver nitrate pencil is employed, in milder forms a 1 or 2 per cent. solution of silver nitrate (painting of the ectropionized lid, or instillation), and the superfluous amount of silver nitrate subsequently removed by a solution of salt. After the cauterization the application of cold for a few hours is advisable. This treatment is continued daily and later, when the secretion ceases, every second day, perhaps with a diluted solution. Complete cure usually requires from three to four weeks. The treatment is the same if the cornea is involved except that greater caution is necessary and especially that all pressure upon the eye must be avoided. To diminish the consequences of a possible perforation the instillation of atropin or physostigmin (eserin), according to the seat of the ulceration, is employed. To enter minutely upon the special treatment of the further complications would lead us too far. In the consideration of these questions we must refer to text-books upon ophthalmology.

### OTHER PATHOGENIC MICROÖRGANISMS

Thus far we have considered only gonorrheal inflammation of the eye in the new-born. Minute bacteriologic investigation of the secretion of the eyes, which has become the rule in the last decades, especially in diseases of the new-born, has revealed that in addition to Neisser's gonococcus not infrequently *other pathogenic microorganisms* (*streptococci*, *staphylococci*, *pneumococci*, *diphtheria bacilli*, *diplobacilli*, *Koch-Weck's bacilli*) have been found in such numbers that the view of a mixed infection is fully justified. Although, according to the result of experimental investigation, we ascribe to the gonococcus in these cases the main part in the deleterious consequences, ophthalmologists have recently published a series of cases in which gonococci were absent (Schmidt-Rimpler, Axenfeld, Chartres, Reyling, Groenow). In some of these cases it was demonstrated that the above-mentioned varieties, either alone or in combination, were the cause of the affection. The rarity with which infectious processes, due to the transmission of such pathogenic germs, are now observed in the mother at the time of delivery, and the greater rarity of the birth of a living child under such pathologic conditions are the reasons that the transmission to the conjunctival sac intra partum is

exceedingly exceptional; therefore it must occur later, in the care of the eyes by the nurse or the mother. The clinical course of these non-gonorrheal affections of the eye shows a number of peculiarities which perhaps may escape the first investigation but become noticeable after prolonged observation. The local irritative phenomena remain within essentially narrow limits. An actual blennorrhic stage of profuse pus secretion is either absent or at most is suggested. The duration of the disease is decidedly shorter and is much more rapidly influenced by proper treatment. A tendency of transmission to the cornea is exceedingly rare. Therefore the *prognosis* with timely treatment is essentially better and the question of prophylaxis is already answered by what has been stated. The *treatment* agrees fundamentally with that of the gonorrheal disease: Disinfection in the florid stage for the discharge of the secretion, and later the employment of caustic measures. With an abbreviation of the first phase, however, the latter treatment begins much earlier.

Finally, there is a third group of inflammations of the eye in the new-born in which the *bacteriologic investigation of the secretion* has given a *negative result*. It is true that microorganisms have been found, but either they were not pathogenic or were present in such small numbers that etiologically they could scarcely be considered. Nevertheless our investigations regarding the pathogenicity of bacteria in the conjunctival sac are not to be looked upon as conclusive. I need only refer to the coli bacteria, the influence of which probably is not quite so benign and, in the new-born particularly, play a great rôle, since they are found in the feces in almost pure culture and an external transmission on the part of the nurse is readily possible. An additional cause of development may be the result of chemical and mechanical lesions. As an example of the former *silver catarrh* has been mentioned. With careless nurses contamination by urine and feces may come under consideration, as well as the entrance of soap while bathing, more or less decomposed milk, etc. A mechanical cause may result from the zeal of the nurse in cleansing the eyes and producing an injury. The infantile eye reacts much more strongly and readily to all of these influences than that of the adult, because the protective mechanism, the ebb and flow of the tears upon opening and closing the eyes, has not yet begun to function and the eyes are usually closed. Clinically these forms as a rule belong to conjunctival catarrh and with the proper treatment they rapidly heal. The prophylaxis consists of the early care and cleanliness of the nurse which has so often been described.

## LOCAL DISEASES

As the diseases of the *mucous membrane of the mouth* and the method of care of the mouth, as well as the *nutrition of the child and the digestive disturbances*, are explicitly described in other parts of this work, among the

local diseases it is only necessary for us to consider *vulvo-vaginitis*, *mastitis*, and *intertrigo*.

**Vulvo-vaginitis.**—This is rare in the first days of life. Its development is almost invariably due to gonorrheal infection, which very exceptionally occurs intra partum, but is usually later, in the care of the child by the nurse. The diagnosis depends upon a thick, purulent secretion, which is soon noticeable, and its bacteriologic constituents. To prevent further distribution energetic measures are necessary provided other infection (eye, mouth) is absent. Therapeutically most authors advise douches with corrosive sublimate, 1 to 5000, or with silver nitrate, 1 to 2 per cent. I have usually limited myself to washing the entrance and the surrounding parts with a corrosive sublimate solution, subsequent dusting with a readily drying powder (dermatol, etc.) and the external application of a quickly absorbent gauze. In this manner the few cases I have seen were speedily cured.

**Mastitis.**—The development of this affection occurs in the new-born similarly as in the lying-in woman. After birth and usually from the third or fourth day the mammary gland in every newly born child is physiologically subject to an increase of function. It enlarges more or less and becomes more distinct, harder, and apparently also more sensitive, exceptionally reaching the size of a walnut or larger. Simultaneously there is a secretion of fluid chemically as well as microscopically identical with mother's milk (witch's milk or colostrum). This physiologic period of lactation remains for a short time at its acme and usually disappears after three or four days, when the breast returns to its previous condition. By frequent squeezing and pressing, as was formerly in vogue, this lactation is prolonged and may be kept up for months. During this physiologic irritative condition the breast is predisposed to infectious processes, just as in adults. Without further inducement streptococci and staphylococci may permeate deeply under the surface by way of the milk passage and cause inflammatory changes and even suppuration. Naturally, this possibility is greatly enhanced the longer the period of lactation, or, if artificially protracted, the more the glandular tissue is disturbed in its activity by compression. Prophylactically, therefore, it is necessary to leave the gland alone and to attend to the necessary cleanliness of the skin and clothing of the child. Direct treatment for the process of lactation, which is entirely physiologic, is unnecessary. With decided redness and swelling, or when the signs of infectious inflammation have appeared, mild inunctions of Credé's ointment or gray salve may relieve the process. But, as in the adult, this rarely succeeds. The application of an ice-bag is not advisable on account of the extreme sensitiveness of the skin of the new-born, particularly to cold. Priessnitz compresses and prompt incision most rapidly retard the inflammatory process. The occurrence of suppuration is not always characterized by a rise in temperature. In severe cases the inflammatory infiltration may pass from the gland to the surrounding tissue (*perimastitis*) and finally reach the axillary cavity. As with every other phlegmon a general sepsis is pos-



sible and cases of this kind have been reported. Furthermore, a circumscribed disease of the gland may eventually lead to contraction, whereby lactation in womanhood readily becomes impossible. By improper incision [it should be radiated, in a line leading away from the nipple.—Editor.] or a rigid retraction of the cicatrix distortion of the nipple may occur, analogous to congenital depressed nipple, and lead to a later incapability of use.

**Intertrigo.**—Intertrigo is a chronic, oozing eczema of the skin in the areas of folds. In addition to the buttocks and the region behind the ear, in children with much adipose tissue the throat, neck, and the flexors of the joints are particularly predisposed. The inflammatory change is not always limited to the folds of the skin, but is often distributed to the surrounding area and may sometimes involve a large portion of the surface of the body. The affected part appears more or less reddened and infiltrated, and the surface oozes or is covered with crusts. Peripherally to the inflamed surface or within it, where the epidermis is still retained, papillary elevations and vesicles filled with serous fluid are found. In the depths of the folds painful fissures appear, and infection causes a development of pustules in the surroundings.

The ultimate cause of intertrigo depends upon external mechanical or chemical irritation. The infantile epidermis after birth is comparatively delicate and but little resistant. [And undergoes a normal desquamation.—Editor.] The region of the buttocks is endangered by reason of the necessary cleansing if it is not carried out with the proper care and caution: The skin is very easily injured by rubbing. Chemical irritation arises in consequence of decomposed urine (infrequent change of the diapers, a close rubber cover), or, in digestive disturbances, from the strongly acid reaction of the diarrhetic discharges. Similar conditions affect the folds and creases upon the neck, nape of the neck, and the joints, except that here the retained secretion of the skin is subject to decomposition. Vomited material which flows down the neck, if not carefully removed, may also cause the condition.

Intertrigo, like every chronic eczema, is merely a local disease. Constitutional symptoms are not present. Nevertheless, we must assume that in the new-born, as in adults, chronic eczema, particularly of the anus and genitalia, in consequence of repeated irritation from contamination with urine is associated with sensitiveness and therefore with increased restlessness, sleeplessness, etc., by which the infantile organism may be debilitated. The danger of septic infection has already been mentioned and its consequences explained.

Prophylactically all irritation must be avoided: therefore scrupulous cleanliness, careful removal of the bath-water and secretion of the skin from the folds, frequent change of the diapers. On account of the constant wetting and contamination the region of the buttocks should be anointed (vaselin) and afterwards powdered. Digestive disturbances and diarrhea are to be prevented by a proper regulation of the diet.

Therapeutically Hebra's treatment by salves and powders is the most effectual. Vaseline is preferable to lanolin, byrolin, etc., on account of its insolubility in water. These substances are used either alone, by inunction of the affected regions and subsequent dusting with zinc oxid and starch, or zinc, bismuth, or diachylon ointments may be employed. In severe cases only the daily application of a 1 or 2 per cent. silver nitrate solution is efficacious. Bathing after each evacuation, as is advised by so many, I cannot sanction after my practical experience, and would rather warn against it. By its frequent repetition the distribution of the eczema is assisted. Much more advantageous is the omission of the bath and cleansing with oil. Naturally, with a simultaneous digestive disturbance and diarrhea the ordinary controlling measures must be employed.

## CONSTITUTIONAL DISEASES

### ICTERUS NEONATORUM

To this point only the general affections of the new-born which owe their origin to some local cause have been considered, but there are also a number of constitutional changes which appear as such from the onset. Most interesting in this connection is *icterus neonatorum*. It occurs in about four-fifths of all infants and, according to Kehrer's compilation, in boys more frequently than girls, in children of primiparae more often than in those of multiparae, in breech presentations more commonly than in head presentations. After a prolonged or complicated labor the child is often decidedly jaundiced. Debilitated children, premature or asphyxiated new-born, are especially predisposed. Jaundice usually appears upon the second day after birth, more rarely upon the third or fourth day. The discoloration, peculiarly, first occurs in the skin; in contrast to stasis icterus of the adult the conjunctivæ are only affected in the severer cases and later. According to Porak, in regard to the intensity of the jaundice three grades may be differentiated: In the first the yellow discoloration is limited to the skin of the face, the chest, and the back; in the second the abdomen and upper portion of the extremities and the conjunctivæ are affected; in the third even the hands and feet are involved. The duration of the jaundice as a rule depends upon its intensity. In the mildest stage, without involvement of the conjunctivæ, it sometimes disappears in three or four days. In the severer grades it may continue until the second or the beginning of the third week. The feces are never decolorized. The urine in the milder forms is not altered but in the severer cases becomes distinctly or even deeply yellow. The biliary coloring matter is found in the urine principally as a color infiltration of cell masses and casts, less often in diffuse solution. According to Cruse and Halberstamm this varying distribution is also noticeable in the course, in that the latter appears later and is the first to disappear. On account of the comparatively slight amount of coloring matter the Gme-

lin reaction is obtained with great difficulty and because of the failure of reaction there has long been a doubt as to the identity with biliary coloring matter. It is most frequently brought about after filtration of a large quantity of urine by the microscopic examination of the residuum. After the appearance of the yellowish brown cell masses the reagent is permitted to penetrate slowly under the cover glass. Hofmeier was the first to practice this tedious method of observation in a large number of new-born. Macrochemically chloroform extraction, perhaps with subsequent evaporation, is necessary (Cruse).

With this simple sketch the characteristics of the clinico-pathologic picture of ordinary infantile jaundice are exhausted. Other deviations are absent. The condition of the pulse corresponds to the norm. Its frequency, inversely to the consequences of stasis icterus in the adult, shows no diminution. The temperature also is uninfluenced. Finally, there is no noticeable disturbance in the subjective condition: The child nurses well, sleeps quietly, and cries no more than others. This general conduct caused many authors to regard jaundice of the new-born as normal, physiologic. On the other hand, the minute investigations of metabolism in the first days of life, such as have been conducted by Porak, Cruse, and especially by Hofmeier, have revealed that jaundiced infants are liable to poorer nutritive conditions and especially the more pronounced the jaundice. Although this result must essentially strengthen the opinion that the condition is pathologic it is of greater importance in etiologic investigation and furnishes for prophylaxis and treatment the support which was formerly lacking.

As icterus neonatorum is not directly dangerous to life and infants never perish from it alone, the autopsy reports are exceedingly scant. This accounts for the tenacity with which the view was long maintained that the yellow discoloration was limited to the skin and conjunctivæ. However, careful investigation of infants who have perished suddenly as the result of accident has in the course of time proved that the internal organs undergo a more or less yellow discoloration. It is a peculiarity that the cartilage, brain, and spinal cord are frequently affected, while in the stasis icterus of adults these regions are not involved. Less apparent, even in the severer forms, at least macroscopically, is the change in the parenchymatous organs—the spleen, kidney, and liver. In the last-named organ Birch-Hirschfeld demonstrated microscopically also in the mild grades a plentiful presence of biliary coloring matter. The icteroid discoloration is particularly distinct in the intima of the vessels, the endocardium, the serous membranes, and in the fluids secreted in these cavities. The mucous membrane of the intestines and the permeability of the ductus choledochus and ductus hepaticus always remain normal in ordinary icterus neonatorum. In the kidneys, in addition to a generally more decided hyperemia, the chief characteristic is the constant finding of uric acid infarcts, with which occasionally there is an accumulation of pigment excretion. The latter, the much disputed bilirubin crystals, considered chemically identical to hematoidin crystals, have been found by Orth and Neumann



also in other tissues and in the blood of icteroid infants who have perished. This observation was for a long time the main support for the theory of a hematogenous origin of biliary coloring matter and therefore of jaundice. Later investigations, however, have furnished positive proof that this only results from a special secondary change after death—a cadaveric condition. C. Ruge found these excretions also in the macerated fetus; Neumann, in children who died immediately after birth, therefore before jaundice could be developed. Finally, Hofmeier was never able to demonstrate such crystals in his numerous examinations of the blood of living icteroid infants nor by prolonged preservation of his preparation could their formation be noted.

In regard to the origin of *icterus neonatorum* there is still a great difference of opinion. The view formerly held of a hematogenous relation must be rejected as untenable, at least in its original sense that either the hemoglobin which is freed by the destruction of red blood corpuscles is changed immediately into biliary coloring matter in the circulation or that this alteration occurs in the small and large blood extravasations in the skin or in the internal organs even after a normal birth. After the investigations of Hayem, Violet, and especially of Hofmeier and Silbermann there can certainly be no doubt in regard to certain changes in the blood, in *icterus neonatorum*, especially that the erythrocytes are destroyed and become free and that hemoglobin thus finds its way into the circulation. Physiologic researches of the last decade, however, have furnished undoubted evidence that even under such conditions the hepatic cells preside over the direct formation of biliary constituents in the secretion of bile, the function of which is not limited to a simple excretion of pre-formed products in the blood. If, therefore, the presence of biliary constituents in the fluids of the tissues and in the urine is to be regarded as a reliable sign of the hepatogenous origin of the existing jaundice, this is especially true of the bile acids, of which another point of formation than that of the hepatic cells is unknown. Birch-Hirschfeld-Hoffmeister and subsequently Halbertsamm succeeded in substantiating this argument in the pericardial fluid. In the icteroid new-born this was found to contain bile acids; in the non-icteroid it was free of bile acids. The latter result should answer Gesner's criticism that in these cases a diffusion of bile occurred post mortem. As the result of this investigation *icterus neonatorum* must be regarded as positively hepatogenous, due to resorption of the biliary constituents formed by the liver cells. The question then remains, to what may this absorption be due. It is obvious that a condition such as occurs in the jaundice of adults—a hindrance in the flow of bile—should first be considered. In this sense Virchow has suggested the possibility of an accumulation of mucus and epithelium in the ductus choledochus. According to Epstein and Cruse the biliary passages, like other organs (for example, the mouth), in consequence of circulatory changes after birth, are subject to a hyperemic, catarrhal, irritative condition with desquamation of epithelium, which causes a constriction or closure of the lumen. Finally, Kehrer considered congenital narrowness of the excretory ducts. All of these attempted explanations are

purely theoretic and lack anatomic foundation. It has already been stated that in ordinary icterus neonatorum the feces retain their color and that signs of obstruction of the flow of bile into the intestine are absent. The greater difficulty with which the bile is discharged by pressure upon the gall-bladder in the autopsies of the new-born in comparison to adults exists to the same extent in icteroid and in non-icteroid subjects. Those cases in which an actual closure of the excretory duct occurred during life are characterized not only by a certain degree of yellow discoloration but also by a greater involvement of the general condition. If such complications occur at all they can play a part only in a very small minority of the cases.

A second group of authors takes the wider view that biliary stasis is due to the circulatory changes to which the liver is subject after birth. It was thought that with the arrest of the placental circulation a stasis of blood developed in the vascular area of the hepatic and portal veins and caused pressure upon the biliary passages. This consideration, originating from West, Hewitt and Weber, which was at first only theoretic, was subsequently found, through the anatomico-pathologic investigations of Birch-Hirschfeld, to be based upon fact. The more or less extensive edema of the periportal connective tissue, demonstrated by this author in icterus neonatorum, was regarded as stasis edema, due to the stoppage of the umbilical vein circulation and an insufficient activity of respiration. Shortly afterward Cohnheim reported a series of observations in ordinary icterus neonatorum in which no edema of Glisson's capsule could be demonstrated. Hofmeier called attention to the fact that in the autopsy findings of Birch-Hirschfeld there were only the extremes of circulatory and respiratory disturbances, from which the infants eventually perished. A generalization of these conditions to the effect that between the normal, vigorously breathing and the asphyxiated child the most manifold intermediate stages exist and that the consequences of deficient respiration become noticeable in proportion to the frequency of icterus neonatorum, therefore in about 80 per cent. of the cases, is absolutely untenable according to the experience of obstetric practice. Finally, Stadelmann utilizes in comparison the sequels in uncompensated valvular lesions of adults. The severest grades of liver stasis only give rise to a very slight icteroid discoloration, and even these cannot be referred to pressure upon the dilated vessels, but, on the contrary, to the interstitial connective tissue proliferation by which the biliary passages are only secondarily compressed. Accordingly an eventual cooperation of blood stasis or of edema of the periportal connective tissue, even in the isolated cases of deficient respiratory activity which Hofmeier himself includes, is not probable. The other foundation for the development of blood stasis in the liver (Silbermann) by a fermentemia resulting from destruction of blood corpuscles does not permit this compression theory as the cause of icterus neonatorum to be recognized and has already been rejected by Stadelmann.

Another group of authors adheres to the causal influence of the circulatory change in the liver after birth, but characterize it as a decrease of blood pres-

sure with a lessening of tension in the capillaries of the hepatic parenchyma, and thus attempts to prove a direct transmission of bile into the blood (Frerichs, Naunyn, Schultze). Opposed to this view is the observation that icterus neonatorum is decidedly increased when the child receives post partum as much of the placental blood as is possible, either by a delayed tying of the cord or by expression of the cord and of the uterus. The increase of the general amount of blood, associated therewith, causes an increase of the general blood pressure, from which the portal vein cannot be excluded. Furthermore, experimental physiology has not been able to determine a decrease of bile secretion even after decided lowering of the blood pressure. Among the local hypotheses that of Quincke must be mentioned. He invokes the well-known biliary circulation in explanation, which in the new-born, in consequence of the direct communication of the portal vein with the vena cava, is diverted by the ductus venosus Arantii. Likewise as in the adult bile from the intestine is absorbed into the blood of the portal vein, and all the more readily in the new-born as the meconium contains a profuse amount of biliary constituents. While in the adult the blood of the portal vein is carried only to the liver and is there freed from deleterious products, in the new-born a portion of the impure blood finds its way through the ductus venosus Arantii directly into the vena cava and thence into the general circulation. If the transference of bile into the fluids of the tissues in this manner causes the ictteroid discoloration, the simultaneous presence of biliary acids in the blood must be responsible for the marked destruction of the erythrocytes. As convincing as this explanation appears at first sight, it leaves completely out of consideration the fact that jaundice does not occur regularly in the new-born and that when it is present its intensity varies considerably. Kehrer has emphasized that asphyxiated infants also, who have discharged their meconium largely intra partum, may become markedly jaundiced and that a coincidence of intensity with a slow or rapid evacuation of meconium cannot be demonstrated. To explain the exceptional cases by the assumption of a special stimulation of absorption on the part of a greater intestinal peristalsis means to base one hypothesis upon another, which will probably satisfy no one.

From this discussion it is obvious that none of the theories is sufficient for all requirements, and our lack of knowledge of the more profound circulatory and secretory processes, that is, the changes in the liver of the new-born, and the hypotheses therefore required, make it doubtful whether this method of search for a local cause will meet with success. Accordingly the investigations which were recently begun are to be welcomed. These take into consideration the entire life—or pathologic picture of the ictteroid new-born so as to determine the difference between their condition and that of the non-ictteroid. Hofmeier was probably the first to enter upon this research and has furnished us valuable evidence. In the first days of life jaundiced infants are subject to a greater loss in weight, due entirely to the consumption of their own body albumin. The slower and later compensation proves that also in subsequent days jaundiced infants possess a slighter energy of



growth. The excretion of urea which under normal circumstances shows a primary rise until the third day and a beginning decrease from the fourth, shows in the first nine days a demonstrable and decided increase. The formation of uric acid shows the same relation: the signs of uric acid infarct and its accompanying phenomenon, albumin in the urine. Hand in hand with these anomalies in metabolism is the change in the blood which, according to the microscopic investigations of Hofmeier and Silbermann is characterized by a destruction of red blood corpuscles and an abundant formation of immature and more resistant forms, and in jaundiced children is even more decided. Hofmeier associates the disturbances of metabolism with the deficient nourishment of the child in the first days, either quantitatively (slight secretion of milk, *primiparæ*) or qualitatively (artificial nutrition), or with its incomplete assimilation (premature birth). In consonance with the law of the physiology of metabolism in such conditions of inanition the albumin of the circulation is first consumed. The change in the composition of the blood plasma, thus brought about, exerts an unfavorable effect upon the blood corpuscles and increases the consequences of any exertion to which they are subject with the onset of extrauterine substantive respiration. The erythrocytes are destroyed even more quickly; the older forms which still exist are destroyed to a greater extent and are substituted by juvenile stronger ones. The hemoglobin which is thus freed in greater amounts is the cause of an increased production of biliary coloring matter in the liver. The excreted bile is richer in pigment and therefore thicker. The normal regulation between formation and flow is therefore the more readily subject to disturbance as the beginning of intestinal digestion establishes a more rapid increase of bile. The consequence is relative insufficiency of the excretory ducts, which in turn leads to stasis and absorption, therefore to jaundice. Finally, there remains the further destruction of the cell stroma of the blood corpuscles which Hofmeier is inclined to associate with the increased production of uric acid and the greater development of the uric acid infarct. Although this theory in its component parts does not entirely remove certain objections, nevertheless the fundamental view that the peculiar conditions of metabolism of the first days of life are in causal connection with the development of *icterus neonatorum* has much in its favor. The other possibility that the anomalies of metabolism are the consequences of jaundice appears exceedingly unlikely, for the reason that, according to general opinion, the amount of absorbed bile is exceedingly slight and the flow of bile into the intestine, as already mentioned, suffers no direct obstruction. Against the assumption of an independent accompanying condition is the regularity and especially the uniformity of the differences in degree. In support of this view it must be accepted that children who externally show no icteroid discoloration, nevertheless have biliary coloring matter in the urine. The greater predisposition also of premature children and those born at term but less well developed becomes more readily comprehensible by a slighter activity or a disproportion between requirement and assimilation. The experience of practice agrees with this.

Since I have ceased to permit the child to hunger until there is an adequate secretion of milk and have seen to it that the child at once receives sufficient food I have much less frequently met with well marked and severe forms of icterus. This difference becomes especially obvious when a wet-nurse is employed from birth.

**Diagnosis.**—The diagnosis of ordinary icterus neonatorum is easily made by inspection; its correctness is confirmed by the additional observation that the child apparently is otherwise normal and by the subsequent disappearance of the discoloration without treatment. In the differential diagnosis of the severer and more protracted forms all of those diseases must be considered *which give rise to demonstrable jaundice in the new-born*. In this connection, among the general diseases septic and pyemic infections have already been mentioned, and among the local diseases, syphilis of the liver, either diffuse, as hepatic cirrhosis, or circumscribed, as gummatous infiltration. To these must be added Buhl's and Winkel's diseases, which will be described more fully later, the sequels of gastro-intestinal catarrh, the so-called catarrhal jaundice, and anomalies of development in the biliary excretory ducts. In addition to the special symptoms it is important to note that children with these conditions, in contrast to icterus neonatorum, present a more or less serious pathologic appearance. Among the malformations we must consider congenital obliteration and complete absence of the larger bile passages. In these conditions the jaundice is especially marked, usually of a greenish-yellow hue. The feces are decolorized, clay-like, whitish. In addition there are the signs of gastro-intestinal catarrh. With increasing emaciation the children perish in a few weeks, exceptionally later. Such examples are quite scarce in the literature (Henoch, Giese, Gessner, etc.).

**Prognosis.**—The prognosis of ordinary icterus neonatorum may generally be regarded as favorable. Nevertheless, in debilitated children the greater consumption of the body albumin and the resulting hyponutrition must be considered. The prognosis of symptomatic jaundice depends upon the causal affection, and therefore, as the condition is usually grave, it must be looked upon as unfavorable, on which account the term "malignant" is used by many authors for this variety.

**Treatment.**—The prophylaxis of ordinary jaundice must be directed primarily toward the exclusion of all conditions which, according to experience, increase the intensity; therefore the complete relief of asphyxia, sufficient warmth, especially of feeble or premature children, and early regulation of the proper diet. These same points are also operative in the therapeutic treatment, of which increase in food and the prompt relief of the consumption of body albumin are primary. Special drug treatment, such as syrup of rhubarb, which was formerly customary, is absolutely unnecessary and is rather to be rejected in view of its tendency to still further decrease the absorption of food.

The prophylaxis and treatment of symptomatic jaundice, in so far as this condition comes into consideration at all, depend upon the causal affection.

## PEMPHIGUS NEONATORUM

We are much better informed regarding the *pathologic importance of pemphigus neonatorum*. In the simple variety vesicles appear suddenly, usually during the night, upon the skin of the trunk, sometimes isolated, at other times quite numerous, in irregular distribution from the size of a lentil to that of a pea, and surrounded by a red areola. A further distribution occurs in crops, similar to its first appearance. Simultaneously the vesicles already present increase in extent to the size of a hazelnut or a walnut, a pigeon's or even a hen's egg, the adjacent vesicles unite and a large complexus may be formed. The shape of the smaller prominences are semiglobular; the larger ones are more oval. In addition the latter show a less degree of fullness, probably in consequence of the more extensive evaporation of water upon the surface. They are no longer tense, but flaccid or even undulating. The contents eventually undergo a qualitative change; at first serous, gray, transparent, the vesicles and blisters become purulent, opaque, and yellow. The final course varies. In some of the vesicles, especially the smaller ones, desiccation occurs: A crust forms which gradually loosens as the epithelium is replaced. Others, particularly the larger ones, rupture and leave a slightly oozing surface over which the skin eventually forms. The affected areas are characterized for some time by a more marked pigmentation, which finally disappears without cicatrization, leaving no trace of the former disease.

As has been stated, the first eruption commonly occurs upon the trunk and most frequently upon the skin of the abdomen, in the vicinity of the navel or toward the inguinal region. In its further distribution the neck and head are involved. The extremities are usually not affected until the process upon the trunk is well developed. The palm of the hand and the sole of the foot are in the majority of cases exempt. Disease in these regions is exceptional. There are a few reports of a vesicle formation upon the mucous membrane of the mouth, especially of the lips, the hard palate, and the tongue.

The extension of the cutaneous process varies greatly in different cases. All of the transitional stages from the appearance of a few isolated vesicles to a more or less dense covering of the larger portion of the surface of the body have been observed.

**Pathologico-anatomically** the localization and the nature of the process correspond closely with ordinary inflammatory vesicle formation. Here also we are concerned with an accumulation of fluid, due to exudation and cell absorption, between the horny layer and the mucous layer of the epidermis, by which the former is raised. An inner partition is present only in the smaller ones; the larger vesicles are of one chamber throughout.

The disease begins usually in the second half of the first week, rarely earlier, exceptionally later. The period of progression seldom extends beyond the end of the second week and the process terminates on an average in three or four weeks.

With moderate vesicle formation the general condition as a rule reveals



no special change. The child eats, sleeps, etc., just as before. Distinct prodromes are absent. The temperature of the body remains normal or shows only a slight rise at the time of the eruption and of the relapses. Large and particularly confluent vesicle formation is more frequently associated with high, occasionally prolonged fever and corresponding constitutional symptoms. Rupture of the vesicles in various areas leads to more or less circumscribed epithelial defects which readily tend to suppuration, especially when the activity of skin formation is deficient. By repeated vesicle formation at the periphery the excoriations may constantly enlarge (*pemphigus foliaceus*). Under such circumstances the nutritive condition suffers. The infant becomes more and more debilitated and may succumb from gradual loss of strength or sudden collapse. These unfavorable cases associated with more or less severe symptoms are exceptional. Although from their course they justify the adjective "malignant," nevertheless, for a separation as a substantive form of disease there are no other points of support.

*Pemphigus Cachecticus*.—Under this term Caillault has described a special variety: in feeble, greatly debilitated children with extensive vesicle eruption upon the trunk, head, and extremities including the palmar and solar surfaces, high fever, marked alteration of the constitutional condition, and diarrhea. The entire course, in contrast to ordinary acute pemphigus neonatorum, has a more chronic character and continues usually for several months. All of these peculiarities are referred to the cachectic condition. With the extensive clinical material of the Berlin Charité Hensch was unable to convince himself of the existence of such an affection except when associated with syphilis. In all cases of this kind which came under observation the autopsy showed definite specific lesions. Other authors coincide with Hensch so that Caillault's findings require further confirmation.

The question now arises whether ordinary non-syphilitic pemphigus neonatorum may also be congenital. Hensch, Wickham, Legg, and von Winkel have reported such observations. The peculiar localization of the vesicular formation, the eruption which continues for several years, as well as the absence of any statement regarding the eventual propagation of the virus to other individuals make the uniformity with the simple form of pemphigus neonatorum exceedingly questionable; therefore a view of this kind is forced, to say the least.

In association with pemphigus neonatorum *furunculosis*, *deep ulcerations*, and *phlegmons* are often observed; therefore affections of a septic nature which are probably to be explained by a secondary infection of the contents of the vesicles or of the epithelial defects. These sequels are of special importance since they may seriously alter a favorable course and cause a lethal termination.

The causal origin of pemphigus neonatorum is still quite obscure. It is positive, however, that the affection occurs by transmission and is therefore of an infectious nature. This is obvious from the occurrence of epidemics in cities (Olshausen-Mekus), in lying-in institutions (Rigby, Hervieux, Ahl-

feld, von Winckel, Faber, Almquist, and others), and finally from the cumulative cases in the clientele of certain midwives (Koch, Dohrn, Zechmeister, Bodenstab). The fact that with strict isolation the further distribution may be arrested is especially important (Moldenhauer, Dohrn). The transmission to nurslings occurs most frequently through a third person; midwife or nurse. The tenacity with which the virus occasionally adheres to these persons without causing infection in them is conspicuous. Even after long suspension of their occupation, disinfection, a change of clothes, etc., the infection is renewed when again engaged in their occupation (Dohrn, Heubner). The direct transmission to persons in contact with the diseased child has also been occasionally reported; mother, wet-nurse, nurse, etc. The most familiar form is the eruption of pemphigus vesicles upon the mammary gland of the nursing mother or wet-nurse. In proportion to the number of infected newborn the disease is decidedly more rare in adults. Their slighter susceptibility is also evinced by a milder intensity of the symptoms which appear.

How the transmission occurs, whether by contact, through the skin, or by way of the air passages, is not yet determined. Older authors incline to the latter, the recent ones to the former explanation. Convincing proofs of both methods apparently are present. Koch observed the first development of pemphigus vesicles on a child upon the genitalia and inner surface of the thigh, to which the nurse had to devote special attention on account of phimosis. A more striking example occurred in an adult who suffered from pemphigus of the face after using the towel that the suspected midwife had employed. On the other hand, Bodenstab reports the appearance of the disease in all persons with the exception of the physician who had entered the room of a diseased child. As proof of contact infection the results of inoculation in healthy persons of the contents of the vesicles is mentioned (Koch, Moldenhauer, Vidal, Blomberg). The vesicular eruption, however, only occurred in the immediate vicinity of the point of inoculation; there was never a wider distribution. It is true these experiments were always made in adults who showed this limitation also with an accidental, natural transmission. But septic infection may lead to similar sequels. One or another of the pathogenic agents, as we shall see, is almost always present in the vesicular contents. Accordingly, with the simple inoculation of the contents of the vesicles the infectiousness is proven but not the method of transmission of pemphigus itself.

The results of bacteriologic investigation of the vesicular contents vary just as greatly. The specific pyogenic organisms—in the milder forms the staphylococcus pyogenes albus and staphylococcus pyogenes aureus and in the severe cases the streptococcus pyogenes—which have been so frequently found, are to-day generally regarded as more or less accidental contaminations which complicate the affection in various ways and are of particular importance among the sequels. Of the others, from the many reports and particularly because of its frequent confirmation a diplococcus which has not yet been exactly classified, appears to be the most reliable and was first discovered

and cultivated from the vesicular contents by Demme, and later by Claesen and Bulloch, and by Whigham from the blood of infants who had succumbed to pemphigus. By subcutaneous injection into mice and guinea-pigs, as well as by rubbing into their skins, this microörganism causes a general infection with a lethal outcome, the blood and organs bacteriologically showing the same coccus. A cutaneous eruption, however, was constantly absent so that the actual pathogenic agent is not proven even here.

In addition to the view, based on the previously mentioned investigations, that pemphigus neonatorum of itself represents a general infection and is further complicated by the addition of septic microörganisms, another theory exists, especially among dermatologists, that there is at first a simple local affection of the skin and that a general disease is brought about only by secondary septic infection of the vesicles and excoriations. The favorable course without any change in the constitutional condition in by far the greater majority of cases, the almost constant combination of severe general disease with local suppuration, render this view by no means unlikely. Riehl was able to demonstrate fungus elements which showed great similarity to trochophyton tonsurans, the generator of herpes tonsurans. The nature and the course of progression of this affection upon the skin it is true require consideration. A stepwise process as from a central point toward the periphery can only be recognized in the vesicular formation, never in the distribution of the cutaneous eruption upon the body. An irregular distribution of the germs by manipulations in the care of the child cannot be invoked in explanation for then the genitalia and their surroundings would have to show the first and most marked distribution.

Lastly, Bohn and Dohrn have ascribed great etiologic importance to the effect of external cutaneous irritants of a mechanical, chemical, or thermic nature. In the first weeks of life, especially during the period of exfoliation, the skin of the child is said to possess a peculiar tendency to react to the formation of vesicles. Although this hypothesis as a general causal explanation has lost much in its original conception by the well-founded assumption of the infectious character of pemphigus neonatorum, the possibility can by no means be rejected that by damage and injury to the skin, no matter of what nature, the accumulation of the specific virus is facilitated; therefore such cutaneous irritation, provided the germs are present, may be the active auxiliary factor.

In the **differential diagnosis** the most important condition is *pemphigus syphiliticus*. Here the localization of the cutaneous eruption is an essential factor. Pemphigus syphiliticus in its first development shows a preference for both the palms of the hands and the soles of the feet, which in the simple form of pemphigus neonatorum are either exempt or are only involved after extensive distribution upon the trunk and extremities. In syphilis the children are commonly born with this cutaneous eruption; in pemphigus this is exceedingly unusual. In addition to the vesicular eruption luetic children usually show other specific changes; macular or papular syphilid, condyloma,



coryza. Furthermore, as a rule, in syphilis the infant is rapidly debilitated and succumbs in a few days. In regard to the composition of the local cutaneous affection it must be mentioned that so long as the syphilitic pustule remains small it presents a flaccid fulness and after rupture leaves an oozing surface which of itself shows no tendency to heal. Finally, the positive result of the anamnesis of the parents and their clinical examination is decisive.

**Prognosis.**—According to the description of the clinical course in the majority of cases the prognosis is favorable, but with an extensive and in particular a large vesicle formation, together with suppuration and an increased temperature, a complication with intestinal catarrh, and the subsequent appearance of furunculosis, ulceration, and phlegmons, the prognosis shows a corresponding change for the worse.

**Treatment.**—In *prophylaxis* the prevention of transmission is the first requirement. Reliable methods to protect the child itself from the disease, in other words to prevent the primary development, are unknown. Nevertheless the proposition to treat every injury of the skin by hot baths, indifferent care of the child, etc., is not advisable even for ordinary sanitary reasons. It is most important to limit the focus of the affection once it has appeared. Strict isolation, not only of the diseased child but of all who come in contact with it, particularly other infants, is primarily essential. The same is true of all the utensils, clothing, bandages, etc., which should be thoroughly disinfected or preferably destroyed by fire. In institutions the greatest danger of propagation is through the nurses; in general practice, by the midwife, and no less so by the physician. Before attending another case or confinement the most careful cleanliness and disinfection of the body as well as of all the clothing is absolutely necessary. Simple washing in water is quite insufficient. The hair and the beard must also receive attention (Heubner). We must never omit to explain to the relatives the readiness of direct and indirect transmission and to insist upon caution.

The *treatment* is expectant. Measures which will positively influence the course are unknown. It is advisable to protect the vesicles so far as possible from mechanical injury and facilitate their desiccation. This caution is particularly necessary in handling the child. The cleansing should not be done by rubbing but by swabbing with cotton or gauze. The drying after the bath is best accomplished by enveloping the child in cotton. When the vesicles are small free dusting with zinc-starch, or zinc-talcum, equal parts. Larger eruptions in which rupture is unavoidable are most advantageously treated like burns of the skin: artificially emptied and covered with a bandage of salve or oil (zinc, boracic acid, bismuth, dermatol salve, oleum lini with aqua calcis). The same treatment is indicated when oozing areas have developed. Some physicians advise compresses with aluminum acetate (1 to 2 per cent.). Others are opposed to the daily bath, but on account of the more ready and careful cleansing by this means I believe it to be especially necessary. As adjuncts to the bath astringent substances (alum, tannin, oak bark), par-

ticularly in severe cases, are very advantageous. To prevent a secondary septic infection (furunculosis, ulceration, phlegmons) antiseptic baths containing potassium permanganate are given daily, or, in addition to the astringent bath, corrosive sublimate (0.3 to 0.5 per bath).

Besides this local treatment the constitutional condition requires consideration; therefore, the best nourishment—from the mother or wet-nurse—and, with threatening collapse, analeptics: wine, brandy with water, etc.

In syphilitic pemphigus the prompt institution of specific treatment is indicated but is rarely capable of preventing the further decline of the usually miserable subject.

### DERMATITIS EXFOLIATIVA

This rare form of disease shows great similarity to the resulting conditions of pemphigus and was described by Ritter toward the end of the seventh decade of the last century. Behrend attempted to prove that this was only a variety of the condition known as *pemphigus foliaceus*. This view, however, could not be maintained. On the contrary, we now generally regard dermatitis exfoliativa as different from pemphigus and as a substantive disease. The principal characteristic is the ready exfoliation of the skin. Similar to the condition in the macerated fetus, large areas may be raised from their substrata or rubbed off, as occurs so readily in a more or less distributed extent in the ordinary care of the infant. Pathologico-anatomically there are reports of an exudation into the lower epithelial layer, the rete Malpighi, therefore between the papillary body and the horny layer. In contrast to pemphigus the amount of the exudate is so slight that as a rule vesicular formation does not occur or is only suggested in isolated areas. The second difference is the local distribution of this change: It does not appear in disseminated foci, as in pemphigus, but begins in one area, usually on the head, and in a short time extends diffusely over the whole or the larger portion of the body.

As a rule the affection begins in the first or second week of life. Other symptoms of characteristic nature, such as fever, etc., are absent. Recovery occurs by gradual regeneration of the epidermis and under favorable circumstances is complete in a few weeks. In debilitated or premature children the **prognosis** is unfavorable, but even then the cause of death is usually due to some complication (pneumonia, digestive disturbance).

The cause of the disease is still obscure. Worthy of note is its predominantly epidemic nature. Ritter was of the opinion that it was the consequence of a septic general infection. Certainly, the decreased frequency upon improvement of the hygienic conditions in the Prague Foundling Asylum was very conspicuous.

The **treatment** is the same as for pemphigus; astringent baths, applications of powder, and above all good nourishment—whenever possible from the breast.

## SCLEREMA NEONATORUM

Much more rare, even in lying-in institutions and foundling asylums, is sclerema neonatorum. The name arises from a peculiar hardening of the subcutaneous cellular tissue with moderate swelling. In a more or less circumscribed area, usually first upon the legs (calf, dorsum of the foot, thigh), but occasionally upon the cheek, the skin becomes hard, tense, as if infiltrated, and can no longer be forced into folds. Upon pressure by the finger no depression remains. The swelling has a soft, bacon-like consistence and at the same time is cool to the touch. The skin appears dry and pale, sometimes of a grayish yellow or gray-blue color, according as to whether or not jaundice is present. This change gradually extends, is distributed to the lower extremity or to the face, and slowly proceeds along the back and to the arms. The anterior surface of the neck and of the chest, the penis, scrotum, vola manus and planta pedis, are usually uninvolved. With this extension active and passive limitation of movement is associated. The extremities become stiff, cadaveric, and this is also true of the countenance. With an involvement of the lips ingestion of food is difficult or impossible. Finally, the infant lies with limbs extended as if made of wood or stone, immovable, and remains so when lifted. The feeble movements of the thorax and occasionally of the lips are the only signs of life. The process does not reach such general distribution in every case. If death occurs early the change may be limited to the legs or to the face alone.

With this change in the body surface a more or less severe alteration of the constitutional condition occurs. The temperature of the body becomes more and more subnormal. Temperatures of from  $34^{\circ}$  to  $30^{\circ}$  C. ( $93.2^{\circ}$  to  $80^{\circ}$  F.) or as low as  $28.5^{\circ}$  and  $22^{\circ}$  C. ( $83.3^{\circ}$  and  $71.6^{\circ}$  F.) have been reported. The rapidity of the pulse decreases to 80 or even 60 per minute. The quality becomes weaker and softer. This coincides with the results of palpation and auscultation of the cardiac contraction, which at last is scarcely perceptible. The respiration usually is slowed, irregular and superficial, but sometimes, in consequence of a complicating pneumonia, it is rapid. Briefly, there is a constantly increasing general debility to which the infant succumbs within one or two weeks, rarely later.

Pathologico-anatomically this cutaneous change is characterized first by a drying and rigidity of the subcutaneous fatty tissue. Upon incision no serous fluid appears nor can it be produced by stroking or pressure. The change has probably a more profound cause in the peculiar composition of the fatty tissue of the new-born, which according to Langer and Knöpfelmacher contains more palmitic and stearic acids and less oleic acid; therefore it may be a direct consequence of the decrease in the body temperature. In consonance with this is the fact that the sclerematous hardening gradually decreases under artificial warming in the incubator. In cases of more protracted course, in addition to the decrease of fatty tissue an atrophic condition of the connective tissue and epithelial constituents of the skin has been



reported. The only organic lesions found were a more or less extensive atelectasis of the lungs with fetal myocarditis, and in older children there was most frequently a gastro-enteritis or pneumonia, rarely nephritis.

The **etiology** of sclerema neonatorum is obscure. We only know that debilitated, premature, or asphyxiated new-born who are poorly nourished and cared for, are predisposed to the disease; furthermore that the lessened activity of the heart plays an important rôle. From the frequent complication with gastro-enteritis, so-called *cholera infantum*, it is proper to consider the results of an immoderate excretion of fluid and therefore an inspissation of the blood as the cause. In regard to pneumonia this explanation is less adequate. Here, as in the other cases, aid is invoked through the assumption of a congenital or acquired cardiac asthenia. There are reports of bacteriologic findings (Schmidt, Aufrecht) which, however, permit of no positive conclusions.

From what has been stated the *prognosis* must necessarily be very unfavorable, especially when the child has been weak from birth. Nevertheless, Soltmann reports recovery in cases in which the disturbance of the general condition had not reached a very great extent and in which the scleremic cutaneous change was but partially developed.

For the **diagnosis** the following is decisive: 1. Characteristic change of the skin—a firm, soft swelling without the ability to raise folds and without pitting upon pressure. 2. The fall in the body temperature. *Differentiologically*, the rigidity of trismus and tetanus differs in that the skin can be pressed into folds and the introduction of the finger into the mouth at once causes muscular contraction.

The objects of **prophylaxis** are apparent: careful relief of the asphyxia and its consequences; maintaining the warmth of feeble and premature children according to the methods already explained, and strengthening by careful regulation of food.

In the **treatment** similar objects are necessary, and, in addition, energetic measures for the dangerous increase of debility and of the cardiac asthenia. The child is not only to be protected against decided loss of body heat but is to be kept thoroughly warm. The external temperature in the incubator, etc., must therefore be higher than is usual—about 44° C. (110.7° F.). A second essential requirement is feeding with breast-milk, either directly from the breast, or, if the infant cannot suckle, by means of a spoon or stomach tube. The administration of Tokay wine (10 to 20 drops every hour) or brandy (3 to 5 drops in water) is serviceable. Soltmann praises the influence of massage and passive movements of the rigid limbs. In addition to stimulation of the local circulation the respiration and with this the cardiac activity are to be encouraged by the crying of the child. Finally, with respiratory difficulties, hot baths and ablutions (44 to 50° C.) are advised but the subsequent cooling brings with it disadvantages.

## EDEMATOUS INFILTRATION

In addition to this actual hardening of the skin by rigidity of the subcutaneous fatty tissue an edematous swelling, *due to serous infiltration of the subcutaneous cellular tissue*, occurs in the new-born, with the same general constitutional symptoms. Both conditions are often designated varieties of sclerema—*sclerema adiposum* and *sclerema oedematosum*. Although there is a certain conformity of the general symptoms which suggests a certain relationship, against this designation is the custom in the pathology of adults of indicating such transudations by the simple word “edema.” Henoeh describes both conditions under the one chapter heading of Sclerema Neonatorum, but he designates as sclerema merely the actual hardening, as edema the serous infiltration. In the latest text-book of Heubner there is absolutely no mention of sclerema oedematosum except as it is separated differentio-diagnostically from edema. With a knowledge of the clinical difference this simplification of the nomenclature appears to me to be well considered, as confusion is prevented by the sharp distinction of edema and sclerema.

The edematous swelling is the same as in adults: The skin presents a uniformly tense, moist, glistening appearance. The color is waxy yellow or somewhat bluish, often slightly marbled. It is cool to the touch and leaves distinct depressions after pressure with the finger. This change usually develops in the calf and distributes itself to the foot, thigh and the genitalia. The trunk, the upper extremities, and the face are rarely affected. Occasionally the edema is limited to the lower portion of the extremities. The swelling is at first doughy, as in adults, and as it progresses it gradually becomes harder until eventually the finger leaves no depression. This condition, in contrast to the scleremic hardening occurs only when the swelling has reached a very high grade, while in sclerema it is observed from the onset. The same is also true of the limitation in movement due to the swelling and tension of the skin.

The general disturbances are the same as those which have already been mentioned in detail in regard to sclerema: low temperature, decrease of cardiac activity, superficial, slow or irregular respiration; briefly, the picture of increasing general weakness.

**Pathologico-anatomically** this cutaneous change is distinctly characterized as a transudative infiltration, as edema. From the cut surface the serous fluid flows profusely. From the subcutaneous cellular tissue this transudation extends more or less to the intermuscular connective tissue. The autopsy findings only in exceptional cases reveal changes in the organs, which according to the experience of later life may be the cause. Demme reports fatal myocarditis, Elsässer and Henoeh cases of nephritis. Under such conditions corresponding exudations are found in the peritoneum, pleura and pericardium. With the exception of gastro-enteritis, however, the primary cause of these diseases, and especially of nephritis, is obscure.

More comprehensible is the pathologic development in congenital endocarditis and disease of the heart—observations which are actual curiosities. In the majority of cases the autopsy shows nothing characteristic. The subjects were usually premature, debilitated infants who merely presented the sequels of insufficient respiration (some degree of pulmonary atelectasis, ecchymoses, hyperemia of the liver, perhaps also effusion into the abdominal cavity) or of cachexia from some other cause (erysipelas, gastro-enteritis, etc.) or of poor nursing or improper nutrition. Here only a congenital or acquired cardiac asthenia can be considered responsible. The seeming incompleteness of this explanation has caused various authors to invoke other factors. Soltmann regards the salient points as a pathologic permeability of the vascular walls and an abnormal composition of the blood. Baginsky is inclined to regard infection as the cause. Naturally, not much is gained from these views. Notwithstanding all of these considerations the profound cause of this edema which thus far is regarded as idiopathic is entirely beyond our knowledge.

The **prognosis**, based on the severe and constant alteration of the general condition, is at least unfavorable. Most children succumb in a few days, especially when complications such as gastro-enteritis and pneumonia exist. Rarely is the advance so moderate as to postpone the lethal outcome for two or three weeks. A few cases of gradual improvement have been reported.

The **diagnosis** is based primarily upon the positive demonstration of cutaneous edema with decided constitutional disturbance. The variation from the scleremic change of the skin has already been mentioned. With erysipelas there is more or less decided elevation of temperature in addition to a marked redness of the diseased cutaneous area. As the result of this affection, particularly in debilitated infants and when there has been poor nourishment and care, the above form of malignant edema may appear either as a complication or sequel. To be strictly differentiated from it is that form of cutaneous thickening, similar to the condition in adults, which occurs in the new-born after erysipelas in consequence of lymph stasis. Apart from the slower development and the milder course the great prostration and its gradual increase is conspicuously absent.

In **prophylaxis** and **treatment** the same rules are to be observed as have been mentioned in the description of sclerema.

Finally, it must be stated that both changes, sclerema and edema, may appear in the same child. Parrot, to whom we owe most of our knowledge of both clinical pictures, has described such cases. On account of the constitutional condition, which is the fundamental cause, the coincidence of these affections is not remarkable.

#### MELÆNA NEONATORUM

It still remains to consider a number of pathologic conditions which possess a special tendency to hemorrhage and hemorrhagic excretions. The



most familiar of these, melaena neonatorum, is characterized by hemorrhages in the gastro-intestinal tract, i. e., by the vomiting of such masses and their passage per anum. Both processes of discharge in varying frequency and quantity may be combined. As a rule the passage of blood by the bowel preponderates. A quantitative limitation also occurs but then principally affects the intestinal passages. The vomited masses frequently consist of more or less altered fluid or coagulated blood; the fecal discharges are usually of a dark, tarry appearance. The latter, in comparison to meconium, are of a more thin, fluid consistence, in consequence of which they are more quickly and thoroughly absorbed by the bed pad. It is true, in early disease this difference may be more or less concealed on account of the admixture of meconium.

Occasionally these hemorrhages occur a few hours after birth, but are most frequent upon the second day. Their appearance after the seventh day is rare. As extreme periods Genrich mentioned the eighteenth and Kundrath the twentieth day post partum.

The disease usually arises suddenly without special prodromes. The evacuations occur at varying intervals and are more or less profuse. The blood loss soon reaches a serious amount: The infant becomes feeble, apathetic; the skin is pale, cool, the pulse accelerated, small, scarcely perceptible, the respirations rapid, superficial. Death sometimes follows with the signs of a hemorrhage after twelve to twenty-four hours. In particularly severe cases the signs of a large internal loss of blood are evident after the first discharge. In some of the cases the hemorrhagic exudation gradually ceases in the course of a day. First, the vomiting stops. The stools show a hemorrhagic consistence for some time and only gradually assume a normal appearance. The infant recovers completely provided that after an interval the hemorrhages do not begin anew, in which case death is almost inevitable.

The mortality varies in different statistical compilations between 35 and 60 per cent., therefore some authors regard a favorable outcome as common, others as rare.

With these bloody evacuations and their sequels—increasing anemia and progressive loss of strength—the constant symptoms of the disease are exhausted. Further investigation is usually without result. The temperature is normal, or, if there is profuse loss of blood, subnormal. Marked tension, distension and sensitiveness in the gastric region or of the abdomen are rare. To suppose the presence of deep ulcerative processes in the gastro-intestinal canal appears to be justified according to some of the autopsy reports (Binz, Rembold), but according to others this is not so.

In addition to this clinically apparent substantive form of melena there is another which occurs *secondarily* in certain general affections. In this connection the predisposition to septic infection and syphilis has already been mentioned. Buhl's disease which is regarded to-day as a modification of general sepsis, must also be mentioned. In these cases the hemorrhagic discharges usually begin somewhat later. The remaining symptom-complex

is correspondingly prolonged owing to the symptoms of the underlying affection. Thus, in the case of sepsis hemorrhages occur in and from other regions, for example, the navel, skin, mucous membranes, etc. Prolonged and intense jaundice with or without cyanosis, together with dropsy, make the likelihood of a general cause obvious. In syphilis the presence of specific changes is conclusive. But lues hemorrhagica is now regarded as a combination of syphilis and sepsis. To this category also belong the acute exanthemata in so far as they affect the new-born, therefore particularly variola. Some authors connect melena with hemophilia, but their evidence is not convincing. Certainly the examples of heredity which have been reported (Rillet and Barthez) can by no means be regarded as unassailable.

Melena belongs to the rarer affections of the new-born. It is impossible to estimate its frequency on account of the great variations in the statistical compilations. The extremes are 1:500 (Hecker, Buhl) and 1:2500-2800 (Spiegelberg, O. Veit). Probably the average is 1:1500, which corresponds most nearly to the experiences of practitioners in general.

Pathologico-anatomically melaena neonatorum, like similar conditions in the adult, cannot be regarded as a substantive disease but merely as a symptom of various pathologic processes and changes. In the constitutional diseases it is a part phenomenon of the hemorrhagic diathesis which is present also in the various organs. The same position is maintained in regard to the local diseases, except that the course of events and the active underlying cause are less obvious. According to the autopsy reports the etiologic significance is most positive in two processes: *circulatory obstruction in the corresponding vascular region, and superficial ulceration*. The former, on account of the thorough transformation which the fetal circulation undergoes after birth, is the more comprehensible. Other especially noticeable disturbances relate to the abdominal vascular supply and have already been detailed in the description of asphyxia. A direct influence, however, can be ascribed to asphyxia only under certain conditions, as asphyxia is comparatively common, melena very rare. Furthermore, children who suffer from melena are seldom asphyxiated when born (Kling, Silbermann). The view that only the severer grades of asphyxia are the cause of melena cannot be proven. The conditions are similar after abnormal and artificially terminated labors, which deserve note from the standpoint of a possible asphyxia. Therefore other auxiliary factors must exist which increase the influence of asphyxia or which are substantively capable of bringing about a similar effect. Among these in particular are injuries to the infant skull. In addition to obstetrical conditions a marked venous hyperemia of the gastro-intestinal canal has been demonstrated at the autopsy in consequence of accidents or crime (Kundrath). Pomorski regards lesions of the vasomotor center as the principal cause, for example, in intracranial extravasations of blood. This condition is said to give rise to hyperemia and hemorrhage in the lungs, in the stomach, and the duodenum, and to ulceration in the last-named area by corrosion on the part of the digestive fluid. He succeeded in strengthening his

opinion by animal experiment, but further confirmation is not at hand. Of other traumata occurring at birth unskillful and rough handling of the infantile body in breech presentations might be serious so far as hyperemia causing hemorrhage from the mucous membrane and submucous hematomata or their rupture are concerned. To a certain extent also those organic changes must be considered which are regarded as responsible for similar conditions in adults. Nieberding reports embryonic defects of the heart and of the large vessels but his conclusions have not been generally accepted. Hepatic diseases in the new-born, as already mentioned, arise principally upon a syphilitic basis, and assume the form of diffuse or miliary interstitial hepatitis. Usually these infants perish soon after birth from other symptoms but the autopsy findings reveal a well-defined hyperemia of the mucous membrane of the gastro-intestinal tract, occasionally also hemorrhages and thromboses in the roots of the portal vein (Kundrath). Similar observations have been made with regard to solitary gummatous focal disease when of great extent and in the immediate vicinity of the larger branches of the portal vein. In this connection the localization in the posterior portion of the lobus quadratus and in the anterior portion of the lobus Spigelli is especially serious. Therefore, signs of circulatory obstruction are present. Early death prevents the development of further sequels. The possibility of an influence upon the further life cannot be excluded. Without special change in the liver the left branch of the portal vein might be endangered by a decided coagulation of blood in the adjacent portion of the umbilical vein, since this coagulation may eventually continue into the portal vein. A large obstructive thrombus in the upper portion of the umbilical vein adjoining the liver is not rare and is due to insufficient derivation of blood, but in the early compensation of this irregularity it usually soon reaches the required limitation. Only after long duration or extensive development of the circulatory disturbance is there an opportunity for further growth, therefore for the consequences which we briefly ascribe to asphyxia.

The question must still be considered whether in the new-born embolism of the gastric and intestinal arteries may occur. The occlusion of small branches often plays an important rôle in the pathogenesis of ulcerations which are peculiar to certain cases of melena (Landau). If this possibility is confirmed the occlusion of larger arterial branches must be considered. Landau regarded as the points of preference the umbilical vein and the ductus Botalli. According to our pathologico-anatomical views the first must be regarded as exceedingly doubtful if not impossible. At events the source must be beyond the lungs. This prerequisite is alone fulfilled by the ductus Botalli. Its strength even in a less congested condition would aid the propagation of a larger blood coagulum, therefore the occlusion of one of the larger branches. Its occlusion by thrombus formation is to-day regarded as pathologic; nevertheless, if the cases of puriform composition of the thrombus are excluded, Rauchfuss noted it in about 0.7 per cent. of his investigations. But in diseases of the heart the emboli distribute themselves within



the region beyond the point of the ductus Botalli, so that the extremities are frequently, the spleen and kidney more rarely, and the mesenteric artery only exceptionally involved. Even if a blood coagulum reaches the celiac artery it is not ordinarily forced into the special vessels of the stomach and of the duodenum. The same relations hold for the ductus Botalli. The lesser distance is without importance and at most excludes the conduction to the head and to the upper extremities. Although by assuming a thrombotic closure of the ductus Botalli it is impossible theoretically to exclude the possibility of embolism, such an occurrence in practice must be regarded as exceedingly rare.

Thus far we have only considered the causes of pure stasis of blood, therefore essentially those cases in which special superficial changes in the gastro-intestinal canal are absent. Upon removal of the hemorrhagic intestinal contents post mortem the mucous membrane appears more or less strongly injected, or after prolonged loss of blood it may be pale and anemic and all the other organs have a decidedly anemic appearance. Here we must accept the view of hemorrhage *per diapedesis* (oozing).

Among the superficial changes in another series of cases *intramucous or submucous extravasations of blood, erosions and ulcerations*, particularly in the stomach and duodenum, have been found. The ulcerations possess all of the properties of the round ulcer of adults: oval or round and sharply limited as if made with a punch, with a funnel-shaped base, sometimes superficial, at other times reaching to the serosa or even perforating this structure. The size varies usually between that of a millet seed and a lentil; but a diameter of 1 to 2 mm. has been reported (Winckel). Usually only a few are present; a multiple development is rare. In the latter case the mucous membrane between is either entirely normal or more or less permeated with blood. Localization within the stomach appears to be somewhat more common than in the duodenum (Anders). Hensch observed an annular ulceration in the lower portion of the esophagus. In the further course, as in the peptic ulcer of adults, there is a possibility of perforation.

The great similarity of the ulcerations in melena with those of the round ulcer of later life leads us to believe that here is the main source of the hemorrhage. In the last decades this idea has gained ground, as in a large number of cases of melena this condition was a positive finding. On the other hand there are reports even of multiple ulcerations in the gastro-intestinal canal without melena or any evidence of hemorrhage in the intestinal contents. It is true such exceptions may be due to the failure of the arterial vessel to rupture. Nevertheless, we must conclude that ulcerations may be the cause of melena, although not necessarily so.

Concerning the development of these ulcerations we have as little knowledge as of round ulcer in the adult. We are dependent more or less upon assumptions, the proof of which is still lacking and exceedingly difficult to establish. Most comprehensible is the nature of the development: that it is due to blood stasis, perhaps under the influence of a fatty degeneration of the

vascular walls which at first causes extravasations of blood. The mucous membrane in the corresponding area thereupon loses its vital resistance to the digestive power of the gastric juice, necrobiosis occurs and thus it is destroyed (Kundrath). After breaking through the protective epithelial cover the further progression is assisted by destruction of the tissue. The development of ulceration in connection with smaller emboli (Landau) is subject in general to the same principle, but is less likely, as already mentioned, if a lesion exists from which it might originate. The observations regarding apparent pyemia in which other diseases of a corresponding focal nature are also present are at most exceptional.

That these ulcerations may develop in extrauterine life, therefore that their onset and distribution occur at the earliest intra partum, coincides with the current view of most authors. Spiegelberg, Bohn and others are of the opinion that with an early disease this period of time is too short for circumscribed ulcerations and that the onset of ulceration is to be antedated to the intrauterine period. Their etiologic attempts at explanation are more or less hypothetical and lack anatomic foundation. Nor is it apparent why even great defects may not arise soon after birth if we remember that the principal instigator, hemorrhagic infiltration, may have already occurred intra partum and that the corrosive effect of the gastric juice requires but little time.

**Diagnosis.**—The diagnosis of melena is naturally based first of all upon the knowledge that the evacuated hemorrhagic masses actually originate from the gastro-intestinal canal. In hemorrhages from the nose, mouth, and pharynx, and even from sucking at the wounded nipple of the mother, hematemesis and entorrhagia have been observed. A careful observation of these changes is therefore always in place. However, such conditions, designated by the term "*melaena spuria*," are rare in the first days of life and are not nearly so liable to cause as rapid and severe conditions of debility.

**Prognosis.**—The prognosis is uncertain. The outcome in the individual case naturally depends upon the strength of the child and particularly upon the amount and duration of the blood loss. If, after forty-eight hours, the excretions show no sign of abatement, the prognosis is correspondingly more grave. According to Silbermann no child in whom hemorrhage has lasted longer than seven days has lived. The prognosis is most unfavorable when the condition is due to pyemia or septic infection. After the arrest of the hemorrhage there is a possibility of serious sequels. Sometimes the infants recover very slowly and for a long time or even permanently are retarded in their physical development. Some authors report a diminished resistance of the digestive tract and a ready tendency to dyspepsia and diarrhea.

**Treatment.**—*Prophylactically* the principal attention must be directed to the prevention of septic infection; notably, careful treatment of the umbilical wound according to the principles which have been explicitly detailed. Whether Landau's propositions for tying the cord are actually of much value is doubtful. In the absence of an urgent reason the cord is to be tied only

after it has ceased to pulsate, or, more correctly, when it collapses, and the ligature is first to be applied close to the body and afterward above.

In **treatment**, combating the hemorrhage and strengthening the general condition are of most importance. The primary requirement is rest, i. e., the infant should be kept in its crib and moved as little as is necessary for its care. The application of cold to the abdomen, especially of an ice bag, advised by many authors, readily aids collapse. Warmth (enveloping in flannel), and the addition of heat (hot water bags, incubator) are of essential influence in the local disease in raising the general condition. The most suitable food, naturally, is breast-milk. When there is much vomiting the frequent administration of cooled milk with a teaspoon is effective. The employment of enemata (ice water, styptic solutions) is not advisable on account of their stimulating peristalsis, especially as the point of bleeding cannot be reached. Among the remedies formerly much employed were liquor ferri sesqui chloridi (one drop every hour in gruel), and ergotin by the mouth or subcutaneously (0.02-0.05 per dose). Recently, by the advice of Carnot, gelatin solutions have been employed for the arrest of hemorrhage (Zuppinger). These are to be given in a 5 to 10 per cent. solution subcutaneously or by the mouth. When employed subcutaneously it is necessary to see that the solution has been recently sterilized.

Threatening collapse calls for the ordinary measures indicated in severe blood loss: low position of the head, envelopment of the extremities, analeptics (wine, brandy, ether).

After the arrest of the hemorrhage good food (mother's milk) and warmth are necessary.

### BUHL'S DISEASE

Another affection in the course of which there is a special tendency to hemorrhage is *acute fatty degeneration of the new-born*, or as it has been called for its discoverer, Buhl's disease. The first designation is based upon the pathologico-anatomical demonstration of extensive parenchymatous inflammation terminating in fatty degeneration of the heart, of the kidney, of the liver, and even of the transversely striped muscles.<sup>1</sup> In this relation the affection shows great similarity to the sequels of poisoning with phosphorus and arsenic. The changes in the organs are sometimes quite similar, at other times varied, some of them already showing advanced fatty degeneration, while others are yet in the stage of parenchymatous inflammation or may even appear normal. The spleen is usually enlarged, softened, occasionally somewhat flabby. The hemorrhages occur as ecchymoses or as large extravasations in the skin, the mucous membranes, the meninges, the pleura,

<sup>1</sup> Even tendons and ligaments. Fifty years ago Dr. Geo. T. Elliott exhibited before the N. Y. Pathological Society many parts of a full-grown baby delivered under peculiar circumstances. It was a breech presentation. Gentle pulling on the feet tore them off. The same occurred as the result of traction on the rest of the lower extremities. The severing of the parts took place in the joints.—EDITOR.



the pericardium, the mediastinal connective tissue, the thymus gland, the peritoneum and the muscles. In the lungs the hemorrhage occasionally assumes the form of a hemorrhagic infarct. The bronchi contain hemorrhagic mucus or pure blood; the alveolar epithelium shows fatty degeneration. The mucous membrane of the gastro-intestinal canal is permeated with ecchymoses and extravasations of blood, its contents are more or less tinged with blood, at other times of the nature that is so characteristic of melena.

The **clinical picture** shows few characteristic phenomena, general symptoms being the rule. Some of the infants are born at full term and apparently normal, others are asphyxiated when born without any demonstrable cause. The respiration is superficial and irregular and reacts to stimulation only transitorily or not at all. The face remains pale and slightly cyanosed. The infant is apathetic, nurses badly, and whimpers rather than cries. Other infants at birth present an entirely normal condition, the debility appearing after a few days. The icteroid discoloration of the skin soon becomes intense and simultaneously cyanosis of the face is more conspicuous. In connection therewith are extravasations of blood and ecchymoses in the skin, the conjunctivæ, and the mucous membrane of the mouth. When the umbilical cord separates there is slight or marked parenchymatous hemorrhage. In addition there may be hemorrhage from the nose, the mouth, hematemesis, and hemorrhagic stools. Occasionally edema is observed, especially in the ankles. With great blood loss dissolution is very rapid and the infant perishes at the end of the first week. In some cases the course is somewhat slower, but death is rarely prolonged beyond a fortnight. The hemorrhages are not always prominent; on the contrary, they may be absent and death occurs with the symptoms of a constantly progressive debility or perhaps may be so sudden that an accident is thought of, for example, suffocation.

Fatty degeneration is generally regarded as an extremely rare disease. We must, however, agree with Runge that some of the cases are obscured by designations such as asthenia, umbilical hemorrhage, melena.

In the **differential diagnosis** the primary debility, the asphyxia, and especially, in contrast to melena, the involvement of various organs and tracts in the hemorrhage, are of value. Pathologico-anatomically the diagnosis can only be made by exclusion. The suspicion of poisoning by arsenic or phosphorus will rarely come into question. Most difficult is the differentiation from general septic infection. In this connection it has already been repeatedly emphasized that diseases of the umbilical wound and of the navel vessels are conclusive. Without a demonstrable primary focus the decision must devolve upon minute bacteriologic investigation. Hecker has already called attention to the similarity of the lesions with those arising from suffocation. This is based particularly upon the cyanosis, the decreased amount of air in the lungs, and the ecchymoses. The microscopic demonstration of more or less extensive lesions in the organ is especially important.

**Etiologically** it is now generally accepted that the condition is due to the consequences of general infection, but its specific nature is still obscure.

For the reason that with the introduction of asepsis and antisepsis the affection has almost entirely disappeared, most authors incline to the view that the pathogenic principle belongs to the group of septic infectious agents.

The **prognosis** is highly unfavorable—fatal. Acute fatty degeneration has always terminated in death.

**Treatment.**—In prophylaxis the prevention of septic infection is of most importance.

The treatment must be entirely symptomatic, namely, control of the hemorrhage from the nose, mouth, and intestinal canal by the employment of styptic remedies, especially of gelatin; improving the general condition by the best possible nourishment (breast-milk), and by heat. Finally, relief of the collapse by analeptics (wine, brandy, ether). All of these measures, however, are usually without result except to temporarily postpone the lethal termination.

### WINCKEL'S DISEASE

This affection, first described by Winckel, in which also the symptom-complex shows a tendency to hemorrhage, is characterized clinically by the appearance of hemoglobinuria. The first recognition of the disease was in the Dresden Obstetric Clinic as an endemic affection and within a month it had attacked 23 newly born infants, i. e., 25.5 per cent. of all the children. The affection usually appeared on the fourth day of life with marked cyanosis and intense jaundice. The color of the urine was bluish brown or olive green and contained large quantities of hemoglobin, some albumin, epithelium from the pelvis of the kidney, and granular casts. The temperature, at first normal or but slightly above ( $38.1^{\circ}\text{ C.}$ — $100.5^{\circ}\text{ F.}$ ), soon became subnormal. The respiration was more or less accelerated but the pulse was seldom rapid. Vomiting or diarrhea occasionally occurred. Upon the removal of a few drops of blood for examination only a thick brownish fluid was discharged from the wound, which under the microscope revealed a decided increase of leukocytes in addition to many pigment granules. Most conspicuous was the rapid loss of strength: the infant soon became apathetic, somnolent, and as a rule perished in thirty hours, occasionally in nine or twelve hours, from convulsions. Of the 23 cases only 4 recovered. At the autopsy the kidneys showed in particular the signs of a hemoglobin infarct and in addition an inflammatory swelling. The cortical substance was broadened, red—or black brown, and permeated with punctiform hemorrhages; in the pyramids this discoloration appeared striated. The uriniferous tubules were filled with granular pigment (hemoglobin). Similar collections of pigment were found in the spleen, which was usually enlarged, hard, and of a deep, dark red color upon the smooth cut surface. Among the other lesions there must be mentioned the wide distribution of punctiform hemorrhages in the internal organs, serous and mucous membranes, swelling of Peyer's plaques and of the mesenteric glands, as well as parenchymatous inflammation, i. e., fatty

degeneration of the myocardium and of the liver. Disease of the navel vessels was found only in one case.

Concerning the question of a causal development, notwithstanding all effort and investigation von Winkel was unable to reach a conclusion. Corresponding observations were later reported in very limited numbers (Sandner, Strelitz, Baginsky, Kamen). The view of most authors is that Winkel's disease depends upon the results of a general infection, the damage being revealed particularly by the destruction of the erythrocytes. The nature of the causative agent, whether a definite microörganism or several, has not yet been determined. Strelitz found streptococci in his case but does not believe they were responsible for the affection. His observation is particularly interesting as the case was a sporadic one, occurring two days after a ritual circumcision. Kamen holds the bacterium coli communis responsible, which he was able to cultivate from the well-water employed in washing the mouth. When this water was no longer used the distribution of the disease ceased. Ahfeld quite properly propounds the question, why the children were not previously affected since the same water had been employed.

**Treatment.**—Therapeutically the same measures are indicated as have been detailed for the treatment of melena.



# THE FEEDING OF CHILDREN

By AD. CZERNY, Breslau

WHEN the feeding of children is made the theme of an article it must be associated with the thought that it differs materially from the nourishment of the adult. Therefore it is possible to consider the technic of feeding only after a consideration of the peculiarities of the child, on the one hand, and, on the other, after we determine the age at which the food of the child and of the adult become equal.

Experience has taught that a food which is nutritive and proper for an adult may produce disease in the first years of life; as the child grows older this condition does not usually occur in the same form and intensity. Many attempts have been made to study the function of the various divisions of the intestinal tract and its adnexa, and, by a comparison of the results, to draw conclusions of their proportions in the child and in the adult. These investigations, although very interesting, have shown merely that in the child there are quantitative but not qualitative differences in the function of the intestinal canal and its adnexa. Examinations of the saliva, the gastric juice, the pancreas, etc., of infants have led to the institution of special dietic rules, wherein the facts that have been obtained are especially considered: but the success which has resulted from the employment of these measures has not been such as to justify us in assuming that the essential object has been attained.

A critical review of all researches relating to the function of the intestinal tract and of its glands would lead to the logical conclusion that the nutrition of the child would differ *only quantitatively* from that of the adult, with the one exception that the food would have to be *liquid* so long as the child is without teeth. The food of an adult, however, would make a child ill, even if administered in a quantity corresponding to the body weight. This proves that the child lacks certain protective measures which have as yet not been demonstrated by research. In the consideration of nutrition we must first ascertain the functions of the intestinal tract and the transformation of food in this area, also the processes of resorption and their extent. Not less important is the destiny of the food in the intermediary metabolism as regards its nutritive effect. The peculiar relation of the latter to nutrition enables us to understand the difference between the infantile organism and that of the adult. In contrast to the processes in the intestinal tract they have been considered but little and studied less. To make these relations clear I must

state, as an example, that for a long time it was thought that the unsatisfactory results from the feeding of cow's milk to children were due to the "difficulty of digestion" of the casein in the milk. The more this subject was investigated the more evident it became that the digestion of casein, if we understand by this merely the processes in the intestinal tract and its absorption, was sufficient in both healthy and sick children. Therefore, insufficient absorption alone cannot be responsible for these poor results. If we inquire how much of the absorbed casein is retained in the organism, i. e., utilized in the growth of the body, it is shown that the retention is by no means parallel to absorption, but that, with very good absorption, there may be insufficient retention.

The insufficient knowledge of the intermediary metabolism has led us astray in our search for the protective agency in the nursing, and the dangers which improper food is liable to produce in children.

The investigations of the *intermediary metabolism* in the child, which will be considered later, have already placed us in a position to choose some foods and determine their amount, but by no means have they brought about a theoretic construction of a food that, in all of its constituents, is suitable alike for healthy and sick children. Especially in this realm experience has proved that theories, apparently well founded, often produce unexpected results, or even quite an opposite effect, when applied to the child.

We are compelled to consider the nutrition of infancy as a separate subject not so much because of our inadequate knowledge of infantile metabolism, but because children suffer more readily and more often from disturbances of nutrition than adults, and that these derangements are dependent upon the nature and quantity of their food.

Clinical experience is the only factor by which we may determine when the food of the child may equal that of the adult. As I shall demonstrate hereafter, we do not at present possess a theoretic foundation for the determination of the age limit. We have contented ourselves with the belief that dentition is the time when the child begins to eat solid food; therefore, at the end of the first year it may be accustomed to the food of adults, although it still receives milk. Such opinions are not scientific and are not even based upon experience. Careful investigations of the functions of the intestinal tract and its adnexa, and of metabolism, have been made only in nurslings and, therefore, do not permit us to determine a period at which the food of the child may equal that of the adult. The special predisposition of the child to nutritive disturbances does not disappear with the termination of the first year of life, as might be assumed from most diet formulas, but at the end of the second year. From that period the digestive disturbances are the same as those of later life.

In designating the end of the second year as the time at which the nutriment of the child and of the adult may be considered in common, I refer only to healthy children. It is obvious that with the appearance of disease this period must be deferred.

Separate consideration has been given to the nutrition of the sick and the well among adults, but, unfortunately, the opposite is the case in the discussions of infant feeding. The most varied foods have been administered alike to healthy and sick children; partly because of the inequality of the food and partly in consequence of the variation in dose, contradictory opinions have been maintained. This has given rise to very dissimilar views in teaching. The lack of discrimination of food for healthy and sick children arises from the fact that the mother's milk is the most suitable food for both the ill and the healthy infant. If the subject of nutrition of children is to be made clear, we must begin by a separate consideration of these two entirely different problems.

In the following I shall treat only of the *nutrition of the healthy child*. The food must be of a nature to permit normal bodily and mental development, in so far as these are dependent upon the food, and to exempt the child from functional disturbances and from diseases due to faulty nutrition. This result can be attained only in children born of middle-aged, healthy parents, carried to full term and free from serious deformities, and who weight at least 3000 grms. at birth. In the following dissertation these are designated normal children. Equal results cannot be attained in weaklings or children of premature birth, or in the offspring of parents who are greatly debilitated, too young, or very old, notwithstanding the same food is administered. This fact must be borne in mind so that the food shall not be considered responsible for unsatisfactory results. Subsequent explanations will refer only to conditions in healthy children.

The best result is likely to be attained if the child is nourished from birth with the mother's milk, provided this is furnished in proper amounts and *at definite times*. Great stress must be laid upon this latter point, because the feeding of the mother's milk does not in itself insure the normal development of the child. Even in the breast-fed child excessive feeding may produce severe diseases which are so common in the infant that is artificially nourished, and which are so difficult to relieve. The younger the child, the greater is this danger. Many are made ill from breast nourishment during the first few days of life or even on the *first day*. This may be avoided if the new-born infant is not fed at all upon the first day, and in the following days food is given but three or four times in twenty-four hours. Afterward, five or six feedings in the course of the day are sufficient. These directions are not given arbitrarily, nor do they follow any authority, but they are derived from observation of normal breast-fed children who have had their food when they wanted it, and not according to the advice of a physician.

The *first day* requires special observation. At this time an important change takes place in the intestinal tract, of which no analogy is observed in later life: The sterile intestinal tract is permeated by microorganisms (Breslau, Escherich, Popow, Schild). As it is impossible for us to know whether we may or may not favor this process of invasion by the administration of food, it is advisable not to feed the child or to give only some in-



different fluid, such as weak tea sweetened with saccharin. Sugar should be avoided as it produces fermentation. It is a noteworthy fact that among savages it is customary, probably the result of experience, not to give the new-born any food for the first twenty-four hours.

Passing over the first days, when healthy children take very few meals spontaneously, it will be found that *five feedings in twenty-four hours*, or at most, six feedings, are sufficient. This leaves intervals of three to five hours between the individual feedings. Therefore the mother or the nurse should be instructed that the child is not to be put to the breast oftener than five or six times in twenty-four hours, the minimum interval in feeding to be three hours. Restlessness between times is the sign of a disturbance of nutrition, and indicates *a prolongation rather than a shortening of the period between the feedings*.

If we follow the foregoing directions we may count upon normal development from breast feeding, and the physician need decide then only how long the infant is to have exclusive breast nourishment. In some cases this period is self-evident, for the secretion of milk is no longer sufficient for the development of the child. It must be stated emphatically that insufficiency of food can never be determined by the arrest or decrease of the weight alone, especially if either of these conditions occur suddenly, for they are usually evidences of a disturbance in nutrition. The only infallible symptom of deficient secretion of milk is an apparent constipation; the child, who has up to this time had one or two yellow stools daily, now has a movement only once in thirty-six or forty-eight hours, and, if the deficiency in food is great, the feces are no longer of the characteristic yellow color, but are tinged a light brown or green. I have designated this *apparent constipation* as there is merely a diminution of the feces because of insufficient food. It is necessary to mention that in this condition the child is not especially restless.

If in the first six months, the breast proves inadequate, an adjunct to the food is indicated. If this occurs later than six months there should be a gradual weaning of the child.

Should the secretion of milk be sufficient in the first six months, the question arises, how long should the child have exclusive breast nourishment. Observation of children who have had exclusive breast feeding for a year or more has shown that this prolonged exclusive nursing does not yield the best results. As a rule, these children are exceedingly fat, so that an equal accumulation of fat later in life must be regarded as pathologic; furthermore they are usually pale. Whether this is the consequence, as is asserted by Bunge, of an insufficient amount of iron in the milk has not been absolutely determined. In these children there is sometimes well-developed rachitis.

Experience has taught that children aged six months, and older, not only assimilate food other than milk but that they develop better than from exclusive breast feeding. *At the end of six months* a normal child should have *additional food*, and in the course of the following three to six months should be weaned. This rule is by no means so invariable that other considera-

tions need not be taken into account. For example, it is not advisable to wean the child during the hot summer, when fresh and pure cow's milk is often difficult to obtain; under such circumstances the period of weaning may be deferred somewhat. On the other hand, another pregnancy—not the appearance of menstruation—may require an earlier abandonment of breast feeding.

If a child aged six months is to be *weaned*, my method is as follows: Once in twenty-four hours substitute a soup for the breast milk. This should consist of a meat broth (from a quarter of a pound of meat), to which sufficient fine grits is added to give the soup a pappy consistence. This soup is to be prepared in the same manner as for adults, but it should contain *no fat* and must be prepared from fresh meat and bones, not from meat extracts. The kind of meat is immaterial; we cannot scientifically demonstrate the advantage of pigeon or of mutton broth over beef as there are no reports regarding the assimilation and absorption of gelatine in the infantile organism. The broth must not be too concentrated, otherwise the child is likely to reject it. As a carbohydrate I prefer grits on account of the stimulating influence of the cellulose upon intestinal peristalsis.

The early addition of carbohydrates is justified, since experience has shown that during the transitional period to a mixed diet they are the most serviceable adjunct to milk.

With this change in the food we may continue breast feeding for several weeks, or for some months if necessary (for example, during the hot summer). The substitute meal of soup is given uninterruptedly throughout the period of weaning. At the beginning of this period a second meal is added which should consist of *animal milk* thickened with some *cereal*, in the same amount and concentration as would be administered to a normal, artificially nourished child of the same age. As I shall refer again to this admixture of milk I will only say here that several days should intervene before there is a further change of food, provided there is no reason for a rapid weaning of the child; in addition, the breast and artificial food must be given alternately, to prevent an accumulation of milk in the breast. Should this condition nevertheless arise, the superfluous milk must be drawn off to prevent a premature arrest of the secretion. It is advisable not to endeavor to increase the weight of the child during the weaning period, and, if this should occur, to limit the amount of food, as the *danger of hypernutrition* is particularly great during this time, although serious consequences may not present themselves until some weeks have elapsed.

In the foregoing I have assumed that the mother is capable of supplying her child with the necessary amount of nourishment. The conditions are analogous if a wet-nurse is employed from the birth of the infant. I shall not discuss all of the requirements which have been stipulated, justly or unjustly, in the choice of a wet-nurse, but shall merely emphasize the fact that we are as yet unaware of variations in the milk of different women which are of importance in the welfare of the infant. I agree with Heubner, who

believes that a chemical examination of the milk is without value in the choice of a wet-nurse.

In German literature the natural feeding of the infant is contrasted with the artificial, that is, with animal milk or other substitute. The French pediatricists have a middle course which they designate "*allaitement mixte*." This term is unknown to German authors, and even the language contains no such expression. Nevertheless, this form of feeding is fully justified, and consists in the *addition of animal milk* when there is an *insufficient supply of breast milk*. It is certain of success, particularly when instituted at birth, and is to be preferred to artificial feeding, which is a hazardous undertaking. The belief of many physicians that the dangers of artificial feeding are now reduced to a minimum has arisen, in my opinion, from the fact that reports have included the favorable results obtained in children who have been fed from the first days or weeks of life with breast milk in combination with artificial nutrition. The greatest danger of artificial feeding arises when this method is instituted from birth—a danger which decreases decidedly if the infant has been fed, even for two or three weeks previously, from the breast. Therefore it is necessary to make every effort to have the mother nourish her child and, when her secretion of milk is inadequate, to institute *allaitement mixte*, and thus avoid an exclusive artificial feeding.

*Allaitement mixte* (mixed feeding) is justified only when an examination of the child reveals that the secretion of milk is actually insufficient. If this method is carried out properly, many a mother will, after a time, be able to nourish her child exclusively, even though lactation began slowly and only gradually increased. For this reason mixed feeding should always be preferred. If we conclude, from the deficient increase in flesh, from the *weight of the child before and after a breast feeding*, or from the scant brown or greenish stools, that the mother's milk is inadequate, we first administer animal milk of the same concentration and amount as would be given to a child of the same age who is artificially nourished. We should, however, observe that the same difficulties must be produced by the bottle from which the infant is fed as would arise from a breast with a meager supply of milk, for otherwise the child would very soon refuse the breast, which cannot satisfy its hunger.

Experience teaches that in many children the addition of a *single artificial meal* is enough for satisfactory growth and development. If it is found, after a few days, that one artificial meal is inadequate, a second, and, if necessary, a third is added. It is well to proceed slowly and cautiously, because the secretion of milk accumulates more rapidly the oftener the child has recourse to the breast. By a too rapid addition of artificial food lactation may cease in consequence of an insufficient withdrawal of the milk. By means of *allaitement mixte* the child may be satisfied with the mother's milk if we substitute one artificial feeding. If a repetition of the substitute meal in the twenty-four hours is necessary, we must alternate between artificial and natural feeding so as to prevent immoderate accumulation of milk in the breast.



As there is no equivalent of the mother's milk for the first six months of life, I prefer to continue the mixed feeding (*allaitement mixte*) for that period, even should the increase of the child's weight be slightly less than the normal.

When it is impossible for the mother to nurse her child, only animal milk can be considered as a **substitute for breast milk** in the first days of life, and particularly the milk of the cow, the goat, the mare and the ass. When I refer to the substitution of animal milk for woman's milk, I ignore the fact that animal milk is usually employed after it has been modified. As a matter of fact but few infants are fed unaltered animal milk. Such attempts have been made upon a large scale only in Milan and Paris, and the results have not sufficed to form a conclusive opinion of the value of the method; nevertheless, in the cities mentioned the feeding of raw milk has not been permanently adopted. In the following I shall refer only to **cow's milk**, because this is the only animal milk of which we have a sufficiently practical experience for its professional employment.

Observations in regard to the nutriment value of *unaltered* cow's milk are insufficient. Cow's milk is always fed to children *after it has been heated*. This produces a change whereby it can no longer be compared with breast milk as a food, even though we may concede that the constituents of both are almost identical.

Lunin has shown by animal experiments that unaltered animal food is of value in nutrition, and, although we are at present unable to explain this fact, it should deter us from altering the physical and chemical constituents of animal milk without some imperative reason.

Nevertheless, we are compelled to boil cow's milk, or at least to heat it, for the purpose of infant feeding, because the milk is liable to become *infected* by microorganisms, which are by no means indifferent, in the course of its transmission from the udder to the child, i. e., in milking, transportation, etc., or the milk itself may contain pathogenic organisms, such as tubercle bacilli. An empiric rule is to *boil the milk* to prevent decomposition; since we have found an explanation of this fact in the advance of bacteriology, and have appreciated its importance, we now speak of "*sterilization*" in infant feeding.

Stress must be laid upon this subject, because there have been many physicians (there are yet a few) who regard *sterilization of cow's milk* in infant feeding as an epoch-making innovation, and believe that all the difficulties of artificial nutrition have been overcome by this process. That the results of artificial feeding are not conspicuously improved by the use of sterilized milk has been attributed by most observers to the incomplete methods of sterilization. In my opinion, the lack of success of sterilization may be referred to the fact that, in former times, children were fed with boiled milk, and, therefore, this later method has not produced any essential change. However, valuable knowledge has been gained from the observation of children who have been brought up by sterilized cow's milk, for we have learned not

to over-estimate the importance of the microorganisms which are present in the milk, as has been done so frequently. If it were true that the dangers of feeding with cow's milk are due alone to the microorganisms which it contains, there would have been, at least in those institutions in which sterilization has been strictly observed, a change in the results of artificial nutrition as remarkable as was observed upon the introduction of antiseptics in surgery. This, however, has not been the case.

After the foregoing remarks a special description of the various methods of sterilization would be superfluous. Therefore I shall merely state that *brief heating (not to exceed ten minutes), followed by rapid cooling, and preservation of the milk at a maximum temperature of 10° C. (50° F.) for not over twenty-four hours, is sufficient for ordinary requirements.* It is much more important to note in how far the cow's milk has been altered by the action of bacteria before it is boiled. An essential requisite is this: The milk which is to be used for infant feeding should be as fresh as possible, and retain its natural alkalinity. This requirement, which has not been sufficiently emphasized, is rarely fulfilled during the summer months in large cities. A test of the alkalinity offers a simple and sufficient control of the purity of the milk, as the acid-producing bacteria develop much more rapidly than the other microorganisms. The degrees of acidity prescribed by the Sanitary Commission in Germany for *marketed milk* are much too high for milk that is to be used for *infant feeding*. The addition of alkalies to compensate for the acid formation which has taken place in the milk has proven *unserviceable* in my experience.

The years during which the sterilization of cow's milk has been considered essential in the technic of infant feeding have further enriched our knowledge. At first the milk was either boiled too long (up to three-quarters of an hour); or, in order to prevent too great a loss to the manufacturer, it was sterilized by repeated heating, sometimes even to temperatures far above 100° C. (212° F.), and then kept for weeks or months as old, *permanent milk* (*Dauermilch*). Experience has proved that children who have been fed for months exclusively with cow's milk prepared in this manner develop Möller-Barlow's disease (infantile scurvy). I have observed a number of children in whom this affection has developed after they were fed with Gärtner's or Backhaus's milk, who recovered from the malady after the substitution of fresh boiled milk. Various pediatricists (v. Ranke, Escherich, and others) have asserted that, notwithstanding an extensive experience in feeding with intensely sterilized milk, they have not seen a case of Möller-Barlow's disease: These reports do not annul my own observations, for there are numerous other investigators (Heubner, v. Starck, and others) who state that the appearance of this disease requires continuous feeding with greatly heated milk for a period of seven to ten months. The number of cases which fulfill this requirement is fortunately not large, as in most children, for various reasons, the food is altered earlier, or some addition is made to the diet, which counteracts this damage.



To avoid the *deleterious effects of cow's milk*, just described, Pasteurization has been proposed, as by this means the most dangerous microorganisms of cow's milk, such as tubercle bacilli, are destroyed. The practical results of this method, which are at hand, do not show a noteworthy influence upon the morbidity and mortality of infants.

In regard to the **quantity of cow's milk** which an infant should receive, and the nature and manner of its preparation, several fundamental principles have been determined from the observation of children fed normally at the breast. My general rule in artificial nutrition is that *from the second week of life a child should have but five meals* in twenty-four hours, that is, there should be intervals of four hours between the feedings, with one or two longer pauses during the night. Upon the first day no nourishment should be given; upon the second day there should be two feedings; upon the third day three feedings, etc.; therefore, only from the fifth day are five meals to be administered. *A child should never be awakened from sleep*, even if a meal is thereby omitted.

This method is based on the fact that the number of meals mentioned corresponds to the number which normal breast children take spontaneously; furthermore, in artificial nutrition a longer time is required to empty the stomach and to develop free hydrochloric acid than in children fed with breast milk. In my experience such a régime offers less danger than the more frequent meals recommended by some specialists, as these cannot be taken by many infants without succeeding harm.

As cow's milk as such is not identical with breast milk in its chemical composition, and, in consequence, an equal amount of these foods does not represent an equal nutritive value, numerous methods have been proposed for the *estimation of the amount of cow's milk required for a child in twenty-four hours*. All such calculations are founded on the amount of breast milk which normal children take spontaneously. That the physician or the mother may decide whether the child requires more or less milk than the average amount which has been proposed is self-evident. But these figures are lacking in other respects. In the first place the basis of these figures has been determined by weighing normal breast fed children, but they by no means represent the necessary or correct quantity of food for the child. Children fed at the breast often become so fat that the same intense accumulation of flesh at any other age would be regarded as pathologic. A nutrition which produces excessive fat can scarcely be regarded as necessary. We must conclude that such an infant receives more nourishment than it requires. Cramer has made mention of this and has accumulated observations which should aid us in the decision of the essentially important question, how little breast milk is necessary for the normal growth and development of a child. His investigations are not sufficiently extensive to solve the problem, but they have shown that the quantity of breast milk which a child requires is much less than that which has heretofore been regarded as a standard, and that, therefore, the amounts of milk previously calculated for infants have been excessive.



This would be immaterial could it be shown that with moderate *hypernutrition with cow's milk* there would be as little apparent damage to the organism as is observed under similar circumstances with breast milk. But such is not the case, for while with the latter there is at most a marked accumulation of fat and a discharge of the superfluous food through the intestine, a hypernutrition with cow's milk usually produces constipation, anemia, muscle weakness, or other obvious symptoms of disease.

Therefore I maintain that a child which must be nourished artificially should receive the smallest quantity of food that is necessary for a proper development of the body. Further, if there is a sufficient increase in the body weight, the amount of food must not be increased as the child advances in age. This latter point usually is not observed, and failure of artificial feeding is the result.

As there are essential differences in the quantitative as well as in the chemical composition of individual constituents, it is not enough to give the same amount of cow's milk as would be consumed by a child at the breast. Some authorities (Budin, Chavane, Rothschild) advise undiluted cow's milk from birth. This method has not been tested sufficiently to permit of its general adoption. Although some infants may thrive with such a diet, we do not know how such feeding would be borne by the majority of children, and since there is no standard dosage of undiluted cow's milk the danger of hypernutrition is great. Under no circumstances can a nursing take the same quantity of cow's milk as of breast milk without any injurious effect.

To avoid hypernutrition with cow's milk, on the one hand, and not to limit the ingestion of water by the necessary restriction of the amount of milk, on the other hand, the *necessity for dilution arises*. Attempts have frequently been made to refer the evil results of feeding with diluted cow's milk to dilution which does not equalize the quantitative variation of breast milk and cow's milk. Investigation of their chemical differences has led to an over-estimation of those differences in many ways, for it is possible for healthy children to thrive with simple dilutions of milk. If it were necessary to calculate fractional percentages of casein, albumin, fat, etc., in the artificial food of a healthy child, then the morbidity and mortality would be decidedly greater than it is in fact, because of the existing difference of opinion of physicians and the circumstances that many children are artificially nourished without professional advice.

A scientific foundation for dilution of milk appeared to be given by an estimation of the nutritive worth of food according to its calory value. I say purposely that it appeared to be given, for food must not only fulfill the heat requirement of the organism, but it has other essential objects which sometimes are forgotten, since we have accustomed ourselves to look upon the calory value of a food as of first importance. We must select a food for the infant which contains the necessary calories for the organism, but this must not be by any means the only or essential factor in our estimation of the food. In the first place, practical experience with nutritive flours has

shown that they do not supply permanently the most important heat-producing constituents of food (carbohydrates, fat, and proteids) in isodynamic amounts without injury to the child; secondly, we have not yet been able to estimate how large a part of the combustive material is lost to the infant organism by insufficient absorption or oxidation, by fermentation in the intestine, etc. Finally, as was mentioned previously, we have no standard figures for the calory requirement of the nursling more than we have for the amounts of food that are necessary.

The results of infant feeding with simply diluted cow's milk are so lacking in uniformity that experimental investigations in this direction have never been abandoned. As it is my object to describe only the feeding of healthy children, it does not appear necessary to enter upon a discussion of the various procedures for the modification of cow's milk which have been suggested, but which for the most part require a more thorough test. I must, however, mention two methods which appear to me to be important.

Gärtner has offered a simple and serviceable solution of the problem of equalizing the amounts of albumin, sugar and fat in cow's milk with those of breast milk by the introduction of his so-called fat-milk (Fettmilch). As experience has already shown, this is particularly useful for infants during the first month; unfortunately, in some cities in Germany, such as Breslau, it is boiled so long as to produce various deleterious effects from its exclusive use, especially Möller-Barlow's disease.

Gärtner devoted special attention to the total albumin contents, but other investigators attempted to produce in cow's milk the proportion of albumin to casein that is found in breast milk. For this purpose they utilized whey instead of water. This theory, introduced by Kehrer, was employed later with Gärtner's centrifugation process by Backhaus, Vigier and Monti. According to experience, normal children become fat in the first month from these mixtures, but whether the whey-milk is more serviceable than Gärtner's fat-milk, and whether the theoretic considerations are correct, remains to be proven.

My method for the artificial nourishment of children from the first week of life is as follows: Cow's milk is first diluted in the proportion of one part milk to two of water. Of this mixture I have 100 c. c. prepared for each meal, to which is added a teaspoonful of milk sugar. For the first period I choose a milk rich in fat or cream<sup>1</sup>, but later a milk poor in fat is preferable. This change in the quality of the milk is due to the great dilution which is made at first. With a milk already poor in fat this dilution would be too great, but in older children such a result need not be feared, since a larger proportion of pure milk is then administered. The milk sugar is added to produce somewhat the normal processes of fermentation in the intestine, as well as to cause a better retention of nitrogen from the small amounts of albumin which are introduced. Heubner, Hoffman and

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<sup>1</sup> This should be obtained by skimming, not by centrifugation, so that the percentage of fat will not be too high.

Soxhlet have attempted to determine the amounts of milk sugar which are necessary to give a diluted milk the same calory value as an equal quantity of breast milk. It is obvious that for each dilution of milk a special addition of milk sugar is necessary if this requirement is to be fulfilled with exactness. However, as has been emphasized, an exact dosage of the individual constituents of the food is not necessary in the nutrition of a normal child, and as the estimation of the calory value of the food by no means represents an absolute method, I believe it to be a sufficient rule that *to each meal a teaspoonful of milk sugar* should be added. I select milk sugar because comprehensive investigations regarding the deleterious effects of various carbohydrates (Keller) have proved that it is most serviceable for children during the first months of life. For the same reason I avoid the employment of other sugars or carbohydrates during the first weeks. The administration of 100 c. c. as an individual meal during this time is sufficient even for robust children, while debilitated children may have a smaller allowance. That from the end of the first week there should be five meals in twenty-four hours has already been emphasized.

As a criterion for the increase of the quantity of milk, I advise that *not before the end of the first year should a child have a liter of milk in twenty-four hours, and never more than this*. The increase should be very gradual, and by this rule every mother will be able to calculate how much milk her child should have at a definite age.

These milk rations, which certainly may appear extraordinarily small, are not sufficient for many children in the first weeks or months of life to produce good nutrition, even with the addition of milk sugar; therefore, from the fourth month, instead of increasing the quantity of milk sugar, I add flour. Careful observations (Gregor), as well as my own experience, have shown that by this early and systematic administration of flour the danger of *rickets* and *scrofula* is not increased, but lessened. The cereals which I employ for this purpose are wheat flour, oat flour and corn starch (Mondamin). I do not agree with many authors, who assert that it more advantageous to employ dextrinous flours. The manufacturing chemist has attempted to solve this requirement by the preparation of nutritive cereals for infants. Here I see but one disadvantage for the children. The more quickly and completely the flour is transferred into sugar the more dangerous it is to the infant. If the rapid and complete resorption of sugar were the necessary factor, then a food consisting of sugar should furnish the best results, for it would save work for the intestine. Experience, however, has taught the opposite: While flour is well borne, sugar has a deleterious effect. My explanation of the favorable influence of flour, in which a slow saccharification in the process of digestion is possible, is that the absorption of water, due to the sugar, occurs more slowly and is more readily compensated for by the organism. The formation of acid by fermentation is more moderate, and at the same time is less effective because it occurs more slowly. In this moderate extent the processes of fermentation are rather calculated to



oppose constipation, which is so liable to occur in exclusive feeding with cow's milk.

As in the case of children nourished at the breast, so also with an artificially fed infant: *After the sixth month we give a daily meal of meat broth with grits.* To prevent an increase of the number of meals the surplus amount of milk is distributed among the other four feedings. Toward the end of the first year, and, in very robust children, even from the tenth month, I permit a few teaspoonfuls of some vegetable, to be given immediately after the broth with grits, such as spinach or carrots, which have been pressed through a sieve. This vegetable diet, which should be begun during the first year, forms a very important constituent in the nutrition of the child in the second year; at that age it is the main source of alkalies and iron, and, as these vegetables are rich in cellulose, they have a favorable effect upon intestinal peristalsis.

As I have already emphasized, one of the most important requirements in artificial nutrition, even of the healthy infant, is to proceed very slowly, and gradually to increase the amounts of food provided. We will know if the food administered is insufficient by the scant increase in weight and by the composition and amount of the feces.

Even in adults we differentiate between *great and small eaters*. This varying apparent requirement of different individuals cannot be referred to heredity but arises gradually from the nature of nutrition in the first and following years of life; either in that the children are given superfluous quantities of food, or that they are given food of little nutritive value, and therefore require larger amounts. My experience has taught that those individuals who are large eaters are feebler in their development and in their resistance to infectious processes than those who are moderately nourished. I must, therefore, add that I determine the *volume of food* by the administration of 100 c.c. to children during the first weeks of life, and increase gradually, until at the end of the first year the quantity of food amounts to about 250 c.c. The average size of the individual meal may be readily determined from these extremes, just as in the composition of food.

Thus far we have been treating of the requirements of healthy children. I have already stated that a strict separation of these from children who are ill is necessary for an understanding of proper and improper nutrition. No one would claim that a food is useless because an adult with gastric disease could not digest it, nor would any one insist that a diet suitable for such a patient is necessary for a healthy person. Only in the nutrition of children is the fact usually overlooked that normal, healthy children differ essentially in their food requirement from those who are ill.

I have, therefore, believed it best throughout to determine the requirement of the healthy child, and must remark again that the nourishment described here is proper only for infants who are born healthy and remain so. It is necessary to know that almost every derangement of nutrition will leave sequels which may persist for a long time after the disappearance of the

obvious gastrointestinal disturbances, and a child thus affected will not thrive from a food that is the same, quantitatively and qualitatively, as that of a child who was never ill. It cannot be emphasized too strongly that the nourishment I have advised is only proper when the infant is always free from gastrointestinal derangements.

It is the opinion of most authors that from the end of the first year a child should gradually have the food of adults. Some physicians advise a trial of meat or eggs even earlier than this. In my judgment these views lack sufficient foundation. The child requires a special food so long as there is a tendency to disturbed nutrition from causes which are apparently insignificant. As experience has taught, this labile equilibrium of the nutritive condition does not cease with the end of the first year, but terminates gradually toward the end of the second. The physician has almost daily opportunity to observe cases in which a good result of nutrition during infancy is more or less destroyed by improper food in the second year of life. It is true that during this time there is a change in the character of the nutritive disturbances, which become less dangerous as the age of the child increases, but only after the end of the second year is there a decided decrease of morbidity due to nutritive disturbances. Therefore, from the end of the second year the nutrition of the child need not differ greatly from that of the adult. It is obvious that this period need not be limited to a definite day or month; it merely represents an average basis for calculation.

In the second year of life milk continues to be the most important constituent of food for children. But to prevent a hypernutrition with this I do not permit the child to have more than a liter of milk daily, divided into four portions, just as I have proposed for the end of the first year. This preventive measure at the same time prohibits an exclusive milk diet. Where the latter method is carried out the child must have much larger quantities of milk to satisfy hunger and to increase the weight. My observation of children who have been nourished with milk exclusively has shown them to be conspicuously pale, with flaccid muscles, to some extent rachitic, and to suffer from constipation. I, therefore, advise against such a régime.

When the amount of milk is limited to one liter it is necessary to give other food. As the albumin and fat requirements of the child are fulfilled by the milk, this addition consists of carbohydrates in the form of vegetables. **The diet of a child in the second year of life** is, therefore, as follows: Five meals in twenty-four hours; two meals to be composed of 250 c.c. milk each, with the addition of zwieback, a roll, or a similar amount of bread without butter, in a quantity sufficient to satisfy the child; two feedings of the same quantity of milk without bread<sup>1</sup>, and the fifth meal to consist of meat broth with grits, rice, barley, noodles, or the like, and followed by vegetables in gradually increasing amounts. This diet may be regarded as very monotonous. In my experience, however, a normal child of this age does not re-

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<sup>1</sup>In the latter half of the second year one of these two meals of milk may be omitted.

quire variation if it has not been taught to expect it; for example, if apple sauce (of which I do not approve on account of the large amount of sugar which it usually contains) is given instead of the vegetable, the child will very frequently decline the vegetable. If such an alternative is omitted the child will enjoy his daily meal of spinach without thought of variation. Numerous other examples might be given.

I must mention here that *infant foods* are unnecessary for healthy children, nor does zwieback prepared by a manufacturer furnish any better results than ordinary rolls.

*In the estimation of the results of nutrition* in the second year the increase of weight, which is so important in the first months of life becomes secondary to other factors which predominate toward the end of the first year, namely, the normal development of the skeleton and of the muscles, of the blood, of the psychic functions, etc.

Unfortunately the literature which relates to *the increase of weight of normal children during the second year of life is meager*. While we estimate a weight of 10 kilograms (22 pounds) as the average for the end of the first year, it is impossible to give a corresponding figure for the end of the second year. We must content ourselves with the knowledge that in the second year the increased weight is proportionately less than in the first, and that it does not show the same regularity.

At the end of the second year a child which has remained healthy is able to assimilate the food of adults and to develop normally under such a diet. If there is to be no mistake later, the amount of milk must be decreased in proportion as the child is given other food. It is most advantageous to limit the milk to a small quantity at breakfast and to omit it for the remainder of the day. In adults it has been determined positively that an exclusive or predominant milk diet will not maintain the body permanently, yet in children beyond two years of age milk is very often given in large quantities with the idea that they are receiving an extraordinarily nutritious food. This view seems incomprehensible, since the majority of physicians, as I have stated, are now of the opinion that a child should be given the diet of adults after the first year.

In passing to a mixed diet in the third year the essential change relates to the animal albumin requirement, which is provided by meat and eggs instead of milk. If it is necessary to caution against professional hypernutrition when the diet is exclusively of milk, this necessity becomes all the more pronounced when albumin is given in the concentrated form of meat and eggs. It has frequently been necessary for me to restrict the administration of meat and eggs to two meals daily, and then only in moderate amounts. On the one hand the albumin requirement of the growing child is not so great as is usually estimated, and on the other hand, we must remember that meat and eggs represent acid foods, and that plenty of vegetables and fruit are necessary to supply a sufficient amount of alkali. This last view governs me, in opposition to other physicians, in the exclusion of meat



and eggs during the first year of life, since experience has shown that young infants are readily injured by acid foods.

In the nutrition of the healthy child I avoid *alcohol*. Although it has been proven that alcoholic combustion takes place in the infantile organism, nevertheless, its use is not justified, because of the resulting deleterious effects and of a plentiful supply of other and more combustible nutritive substances.

To enter upon the individual points of diet in the *third year* appears to me to be superfluous, as the same fundamental rules apply to children as to adults. I must emphasize only one point: While I regard five meals in twenty-four hours as necessary and advantageous during the first two years of life, I believe that it is better to diminish the number of meals to three after the end of the second year. As a matter of fact three meals a day is the custom of many countries, but only with the lapse of time, and by the combined action of many physicians, will we be able to overcome the conventional five meals a day in Germany. Although the habit of numerous meals may not be harmful, nevertheless it often causes loss of appetite, capriciousness, and a tendency to digestive disturbances.

I must state in conclusion that while I am perfectly aware that the foregoing is not the only method by which a normal child may grow and flourish, it is my duty to advise only such a method as will furnish a most certain immunity from disease. It is not my object to bring up children who are fat-wonders, but to create in them a healthy color and a vigorous mind and body, to insure for them a good appetite and proper sleep, and to aid them to resist disease; briefly, to develop *healthy children*.

# THE MOST COMMON INFECTIONS OF THE ORAL MUCOUS MEMBRANE IN CHILDREN

By A. MONTI, VIENNA

By means of the atmospheric air, the food, and other substances which come in contact with the oral mucous membrane of children, for example, by the ingestion of food and in cleansing the mouth, quite a number of saprophytic and pathogenic microorganisms gain entrance by which sucking, chewing, etc., may produce an excoriation of the mucous membrane, and, together with the food residua, present a suitable culture medium for the further development of these bacteria.

These changes vary in that saprophytic microbes alone, or in combination with pathogenic bacteria, exert their influence. We will, therefore, first discuss those changes of the oral mucous membrane which are due to saprophytes, and then pass to a consideration of diseases which are the effect of pathogenic bacteria.

## I. CHANGES OF THE ORAL MUCOUS MEMBRANE DUE TO SAPROPHYTIC MICROBES

Miller, in his pioneer investigations, has isolated twenty-eight varieties of saprophytic bacteria from the cavity of the mouth. For clinical purposes a discussion of the changes, and a description of only the most frequent varieties which appear in this region, are sufficient.

**Leptothrix buccalis.**—The *leptothrix buccalis* is the most common microorganism found in the oral cavity of children.

Microscopic examination of the white coating of the teeth, which has been spread in thin layers with distilled water, reveals small or larger clumps consisting of minute round bodies, at the borders of which there are many thin, curved strands. These round bodies are the so-called matrices of the *leptothrix buccalis*. The strands vary in length and are 0.5 to 0.8 $\mu$  in breadth, curved and tortuous, detached, immotile.

If a small quantity of this white coating is combined with a drop of lactic acid in a solution of potassium iodid, and placed under a 1/350th magnification, the greater portion of this coating is seen to consist of epithelial cells, clumps of micrococci and rod- and strand-shaped structures of a yellowish or yellow tint.

**Bacillus maximus buccalis.**—In addition to the variety of leptothrix just mentioned we also find the *bacillus maximus buccalis*, so designated by Miller, which is arranged in strands, or, more frequently, in distinct chains or rods, 30 to 150 $\mu$  in length, running parallel or crossing one another, with regular contours and usually with equally thickened ends. This bacterium stains blue with iodine, but not all the cells give the same reaction. Many strands stain uniformly, others show areas of brownish violet color.

**Iodococcus buccalis.**—Another variety appearing in the cavity of the mouth has been designated *iodococcus buccalis* (Miller). This microorganism appears in chains of 4 to 10 cells. These chains have a sheath wherein the cells are shaped like flat discs or four-sided bodies, sometimes with rounded corners. The sheaths do not stain with iodine or at most they present a faint yellow color under prolonged exposure; the cells, however, are always stained deep blue or violet.

The *fungi above-mentioned* adhere also to the tongue, particularly to the filiform papillae and in the grooves between them.

Their culture media are desquamated epithelium and decomposed remains of food.

**Mould Fungi.**—In addition to leptothrix other saprophytic microbes, such as the numerous varieties of *mould fungi*, may be found in the mouth.

The most common are small-celled sarcinæ, which rapidly multiply in the oral cavity, and also appear upon the uvula, the palatine arch, etc., as a whitish area resembling thrush. They are found loose in small clumps between the epithelia of the tongue, of the mouth, and of the pharynx, or are arranged in clusters at the border of the epithelium. They may even form a coating.

It is certain that the saliva has an antiseptic effect upon these saprophytes, and with a sufficient normal secretion these bacteria are not capable of producing essential disturbances in the mouth. But in children under two years of age, in whom the production and composition of the saliva is not yet completely developed, or in individuals in whom the salivary secretion has undergone a pathologic change due to general nutritive disturbances, these saprophytes may give rise to the well known local and chemical changes in the oral cavity which are designated "catarrhal stomatitis."

**Catarrhal Stomatitis.**—Because of the deficient function of the salivary glands in the new-born, catarrhal stomatitis is an almost constant lesion during the first days of life. A more or less conspicuous redness develops in the mucous membrane of the mouth, which leads to a partial desquamation of the epithelium.

The oral catarrh of the new-born reaches its acme in four to six days, according to the action of the saprophytes, and as a rule disappears gradually in the course of eight to fourteen days; with improper care of the mouth, however, it may continue longer. Where the mucous membrane is tense, especially on the palate and on the tongue, large areas of epithelium may desquamate, leaving excoriations and small extravasations of blood.



In catarrhal stomatitis of the new-born the child sucks painfully and with difficulty, and as a consequence less nourishment is taken. This diminished feeding may readily lessen the secretion of breast-milk and the contact of the oral secretion—which contains the saprophytes—with the delicate nipples produces reddening, irritation, and excoriation.

Catarrhal stomatitis usually results in recovery without special treatment.

If, however, pathogenic bacteria gain entrance to the mucous membrane during the course of this malady, they find a suitable soil for the production of pathologic changes, which will be described later.

In *nurslings* and *older children* these saprophytes cause local changes, especially if the function of the oral mucous membrane has undergone damage from insufficient care of the mouth, from intercurrent febrile affections, or from inflammatory processes of neighboring organs, whereby a favorable culture medium is offered for the microbes. The phenomena are the same as in the new-born: redness, swelling, desquamating epithelium, and saprophytes which may be demonstrated microscopically.

By means of, or even without, this local action, and whether or not the oral cavity is cleansed, these saprophytic bacteria may produce *acid fermentation* in the food residua in the mouth: lactic acid-, butyric acid- or acetic acid-fermentation. Notwithstanding the normal appearance of the mucous membrane, children exude an offensive *acid odor from the mouth*, particularly when the tongue is slightly coated, and the reaction of the secretion in this region is acid. This odor is particularly noticeable in the morning and is very often explained incorrectly. In consequence of this fermentative process the child shows a certain aversion to food and the cause is erroneously referred to as gastric catarrh. Upon close examination of such cases, however, we find a normal function of the stomach, and proper disinfection of the oral cavity soon disperses the trouble.

In addition, these saprophytic bacteria may exert a diastatic action upon the carbohydrates of the food. The children then complain of a *sweet taste*, reject certain foods, or take very little nourishment, although the digestive organs may be intact.

Usually these bacteria have a decomposing action upon the albumin substances and fats in the remains of food in the oral cavity which changes them first into peptones and finally produces putrefaction. These patients often emit such a strong *putrid odor* from the mouth that even strangers note it. In this condition the tongue is furred and there is a thin white coating upon the gum. These symptoms are very often incorrectly referred to digestive disturbances.

Here also a proper disinfection of the oral cavity rapidly relieves the trouble.

So long as the foregoing changes are not extensive the symptoms are local; if, however, the lesions become marked by the action of these saprophytes, as may easily occur with improper care of the mouth, they may lead to infection of other organs: to infection of adjacent areas if the teeth are

carious, to disease of the respiratory passages (putrid bronchitis, etc.) by the inspiration of putrefactive bacteria, to infection of the digestive organs by deglutition of the bacteria of fermentation and their toxins. Large quantities of gas may accumulate which give rise to various disturbances. Such a large quantity of alkaline products may be formed by decomposition of proteid substances that when they reach the stomach there may be a neutralization of the hydrochloric acid. Absorption of the toxins may also cause disease.

However, our knowledge of these sources of infection at present is so slight that an accurate description is impossible. We must limit ourselves in this article to a consideration of the control of the saprophytes, and insist upon thorough cleanliness of the mouth in all children.

## II. INFECTIONS DUE TO PATHOGENIC BACTERIA

When pathogenic bacteria gain entrance to an altered or excoriated mucous membrane, brought about by the action of saprophytes, they usually produce pathologic changes, whereas they are without effect upon the normal mucous membrane.

**Oidium albicans** (*Thrush; sprue; muguet; soor*).—In the new-born the *oidium albicans* often causes a peculiar infection of the mucous membrane. The germs of thrush commonly find their way from without to the oral mucosa by means of the inspired air, or by adherence to substances which come into contact with the oral mucous membrane of the child (the nipple, substances with which the mouth is cleansed, in artificially-fed children the rubber nipple, etc.). It is also believed that the *oidium albicans* collects and multiplies upon acid—fermentating residua of food—substances containing sugar and starch—and are thus introduced with the nutriment.

These spores may be present in the secretion of the oral mucous membrane even in healthy children, but here they do not find a suitable soil and are harmless.

The *oidium albicans* adheres and proliferates only upon a mucous membrane which is irritated and excoriated; it then forms disseminated, ring-shaped, punctiform white deposits, preferably upon the papillæ of the tongue, from whence it attacks the mucous membrane of the cheek and of the palate, and may implicate the entire mucosa of the mouth.

*Microscopic examination* of these punctiform deposits reveals numerous strands and spores of the *oidium albicans*, and in addition various mould fungi, epithelium, etc.

These deposits rapidly multiply and enlarge, and when the disease is further advanced they form a white membrane-like covering of the mucous membrane which is especially marked upon the tongue. If the formation is extensive, similar coatings may appear on the lips, cheek, palate, and the gums, so that the entire oral cavity may seem to be covered by layers of the *oidium albicans*.

*This bacterium is firmly adherent to its foundation* and in well-marked cases presents a dirty white, tenacious, crust-like mass. The underlying *mucous membrane is reddened*, loose, and bleeds readily; the papillæ of the tongue are red and here and there have lost their epithelium. Those areas of the mucous membrane which are free of the *oidium albicans* often appear to be in a state of *inflammatory irritation*. When the disease is prolonged this inflammatory reddening of the mucous membrane and swelling of the papillæ of the tongue decrease and the epithelium presents a glistening appearance.

The *oidium albicans* occasionally develops also upon the posterior wall of the pharynx, where it forms small, firmly adherent points; it may even find its way into the esophagus, although here it never reaches a great intensity.

Clumps of the *oidium albicans* may sometimes be found in the *gastric juice*, but firmly adherent vegetations upon the gastric mucous membrane have not been demonstrated. According to Grawitz the yeast organisms found in the gastric juice of children suffering from the effects of the *oidium albicans* are only peculiar gonidial forms of this parasitic fungi which develop into long strands in the acid gastric juice. The formation of such gonidia in the stomach causes a functional disturbance, such as *dyspepsia* which is a frequent accompaniment of thrush. The passage of these gonidia through the intestine with the dejecta may, on account of the increased acidity of the feces, produce an irritation of the skin *surrounding the anus*, and it is certain that the *erythema and eczema* in this region, so frequently observed in children who are affected by thrush, owe their origin to this circumstance.

The distribution of thrush to the *nose and larynx* has been much questioned, and is undoubtedly a rare occurrence.

Some authors believe that the *oidium albicans* is capable of entering the vessels and causing *metastasis*; for example, Zenker mentions thrush metastasis in the brain, Brindeau in the parotid gland with abscess formation.

The *symptoms* produced by the *oidium albicans* vary according to the distribution. Small islands upon the tongue and upon the mucous membrane of the cheek cause but little inconvenience. With a greater dissemination sucking becomes difficult and painful, the child cries, and there may even be a rise of temperature, loss of sleep with accompanying unrest, and digestive disturbance—*dyspepsia*, intestinal catarrh; when the soft palate and the posterior pharyngeal wall are attacked, deglutition is difficult. If the disease is prolonged the growth of the child is retarded, and on account of the insufficient ingestion of food there is rapid emaciation, loss of sleep, and pain. Under such circumstances there may be a fatal termination, naturally not as a direct consequence of the thrush affection, but as the result of complications, such as acute gastric and intestinal catarrh, etc. In those rare cases wherein the larynx is implicated there may be hoarseness, cough, croupy respiration, and attacks of suffocation.

In children who are debilitated or who have insufficient attention relapses are quite possible.



*Diagnosis.*—Diagnosis with the aid of the microscope is easy. In the white coating will be found a dense mass of strands, round cells, epithelium, mucus, corpuscles, micrococci and milk globules, and, if detachment is difficult, blood-corpuscles may also be apparent in the microscopic specimen.

The thrush strands have a double contour, are transparent and colorless, either unbroken and solid, or elongated, separated from one another by sheaths of colorless or pale yellow links in which occasionally small round cavities may be distinguished. The individual strands also show thready processes (gonidia), which form an irregular net-work. At the end of the thread we occasionally see a clubbed swelling, the so-called spores; these are round, quite large, strongly refractive, pale or pale yellow, and are isolated, or united in irregular clumps of two, three, or more.

If the affection is prolonged the thrush masses consist for the most part of spores, which contain numerous microörganisms, but few or no strands.

The histologic examination of the oral mucous membrane which is covered by the parasite reveals that these have layered themselves *upon the epithelial coating*, and that they collect principally in the grooves between the papillæ. Isolated mycelial strands and spores may penetrate more deeply between the epithelial cells.

The diagnosis may also be made by *culture methods*. The parasite grows best in feebly acid or sugar-containing gelatin, and upon potatoes. By repeated inoculation pure cultures may be obtained.

These measures easily determine the *diagnosis of thrush* and error would seem impossible. As the microscopic examination, and even the culture process, can readily be carried out by any educated physician, a discussion of the differential diagnosis from other diseases of the oral mucous membrane will be omitted.

*Prognosis.*—In healthy children the disease is readily amenable to treatment, notwithstanding its intensity and distribution, particularly if the child is nourished with breast-milk and is protected by favorable hygienic surroundings. Only in debilitated children, nourished artificially with various infant foods, condensed milk, etc., who are suffering from gastric or intestinal disease, is the affection likely to be severe. In such cases the prognosis depends upon the nutrition of the child, the severity of the underlying gastrointestinal affection, and the duration of the disease.

**Stomatitis gonorrhoeica.**—Another infection of the oral mucous membrane by pathogenic bacteria has recently been recognized,—that due to *gonococci*.

In rare instances the oral cavity of the child at birth is infected by the transmission of *gonorrheal secretion* from the vagina and vulva of the mother, whereby a peculiar form of stomatitis arises which has been designated *stomatitis gonorrhoeica*.

In this infection the entire oral mucous membrane is reddened, swollen, permeated at individual points by pus. *Gonococci* may be demonstrated and cultivated from the secretion of the mucous membrane.

This form of stomatitis usually develops slowly and becomes noticeable only after the fourth day from birth.

The staphylococci and streptococci which are simultaneously present in the secretions of the vulva produce an extraordinarily painful and stubborn purulent infiltration of the soft palate and of the tongue.

Severe catarrhal stomatitis in the new-born, running its course with purulent infiltration, should awaken the suspicion of gonococcus infection, and this condition should receive primary consideration.

**Staphylococci and Streptococci.**—A more frequent infection of the mucous membrane of the mouth is by *staphylococci* and *streptococci*.

The transmission of these organisms to the mucous membrane of a healthy child is usually without effect, but if the mucosa is inflamed as a result of mechanical injury or the action of saprophytes, these microörganisms find a suitable soil for infection which runs a peculiar course.

Redness and swelling of the oral mucous membrane suddenly develop, particularly at those points at which there has been previous damage; in consequence sucking is difficult and the child becomes very restless.

According to the condition of the mucosa prior to infection the lesions occur variously on the lips, the tongue, the soft palate, etc. The soft palate in particular is often affected, while the mucous membrane of the hard palate remains normal,—a coincidence which is explained by the fact that the epithelium of the soft palate is often scratched by unskillful cleansing of the mouth.

This affection is usually combined with a rise in temperature, seldom greater than 39°-40° C. (102.2° to 104° F.) which is more or less elevated according to the distribution of the process.

Twenty-four to forty-eight hours after infection the implicated areas of the mucous membrane are infiltrated and show a purulent covering; in the new-born this condition is most common on the soft palate and palatine arch; occasionally the lips, the tongue, the hard palate and the posterior pharyngeal wall are infected. In severe cases the entire oral mucosa may be implicated.

Microscopic examination reveals staphylococci and streptococci, which are readily susceptible to cultivation.

The *course* of the infection is as follows: After a few days the purulent infiltration reaches its acme; the mucous membrane becomes pale, the febrile phenomena are less intense, the purulent infiltration desquamates and the excoriated areas cicatrize. This process advances slowly and may last several days or even weeks.

In severe cases the infection may involve the underlying structures and thus deep ulcerations may arise in different areas of the mucosa, most frequently upon the borders of the jaw, the lips, the frenum of the tongue, and even upon the hard or the soft palate. When such a condition is present there is usually an evening febrile exacerbation, the child becomes restless, and nursing is difficult. If cicatrization is delayed the child is debilitated

and an infection of the gastric and intestinal mucosa, septic intestinal disease, or the symptoms of general sepsis may appear. The purulent infiltration of the mucous membrane by the entrance of cocci leads to more or less swelling of the adjacent lymph glands. The skin may become infected from the flow of saliva and result in phlegmonous inflammations upon the neck, the face, etc.

This infection of the oral mucous membrane is sometimes complicated with thrush.

*Prognosis.*—The prognosis is dependent upon the intensity and extent of the process. With widely distributed ulceration the disease is tenacious. There may be septic infection of the neighboring organs, of the nose or the intestines, or of the entire mucous membrane, and there may be a lethal outcome due to general sepsis.

**Stomatitis aphthosa.**—Staphylococci may lead to a typical infection of the oral mucous membrane which has been designated *stomatitis aphthosa*, wherein a coagulated mass is deposited in the upper layers of the mucosa.

This affection is observed in children before and following dentition; it is rare before this period, is frequent during teething, and reaches its acme between the first and third years of life.

In the intact mucous membrane the staphylococci are inactive; but if the mucosa has been damaged by accumulation of saprophytes or has been irritated by food or by chemical or mechanical means, the microbes find a suitable point of attack. It is assumed that the ingestion of certain foods and drinks, such as sharp cheese, fruits in process of fermentation, pungent spices, very cold or hot fluids, and mineral or organic acids, may damage the epithelial layer of the oral organs to such an extent as to permit infection by staphylococci. It has also been maintained that the ingestion of milk obtained from animals infected by *foot-and-mouth disease* is capable of producing a similar affection.

*Aphthous stomatitis* may be the sequel of other infectious diseases, provided saprophytes have prepared a soil suitable for its development.

That stomatitis aphthosa is an infection due to *staphylococci* is demonstrated by the clinical phenomena, such as typical fever, implication of the lymph glands, and the contagiousness of the disease.

From the foregoing it is obvious that two factors are necessary for the development of aphthous stomatitis: 1. A disease of the oral mucous membrane which runs its course with increased secretion and with an elevation of temperature; 2. that staphylococci find their way to this altered mucosa, which may then be demonstrated as the pathogenic agent.

In consequence of staphylococcus infection, isolated inflammatory areas of small and usually rounded contour appear upon the mucous membrane, with redness and swelling of the surrounding epithelium. In the further course the inflammation is distributed over the entire mucosa, so that the lips, the gums and cheeks, the soft and hard palate, and the tongue are intensely red and swollen.



In the next day or two an exudate is formed *under the epithelium* in the areas originally reddened and inflamed, which, at various places upon the oral mucosa, produces firmly adherent *flakes* which are round or elongated and of a pale color in the thin layers or of a yellowish tint where they are thicker. Closer examination with the aid of the microscope reveals an amorphous mass composed of epithelial cells, fibrin in small granules, more or less numerous young cells, but principally of cocci. The saliva shows an increase of epithelial cells and numerous leukocytes.

The affection reaches its acme in two or three days. The mucous membrane then begins to fade; the exudate is gradually surrounded by a red areola which becomes quite distinct, and by resorption the superficial layers decrease in size and finally disappear under the formation of new epithelium.

If the exudation is profuse the epithelium is raised and may be desquamated. Even in such areas only a white speck remains, which in time disappears.

Aphthous stomatitis *never leaves cicatrices*.

The disease runs its course with *fever*. Simultaneously with the redness and swelling there is a rise of temperature in mild cases to 38° C. (101° F.), and in severe affections it may reach 40° C. (104° F.).

After exudation is completed, which occurs in two or three days, the temperature falls and during resorption and elimination of the exudate there are at most slight evening exacerbations. Only with renewed exudation is there a recurrence of fever.

It will therefore be readily observed that *aphthous stomatitis* usually runs the following *course*: First, disseminated or cumulated redness and swelling appear on different portions of the oral mucous membrane with salivation and fever. Later the inflammation distributes itself to the entire mucosa of the mouth. The child refuses food because of pain; mastication is especially troublesome and may cause hemorrhage of the mucous membrane. As the result of pain and of fever the child becomes restless, sleepless, and cries.

In the two succeeding days a profuse grayish-white exudate is formed upon the originally reddened areas, with enlargement of the submaxillary glands. By confluence of this exudate larger areas may reveal the same appearance; such a condition is especially common at the tip of the tongue.

The subsequent course depends upon the intensity of the infection. If the process is limited to the tongue, the cheeks and the lips, in three or four days the redness and swelling of the mucous membrane and the salivation decrease, and the exudates disappear in the same sequence in which they developed. At the same time there is amelioration of the functional disturbance and the child again takes food.

In rare instances the exudate may be deposited upon the epiglottis and at the entrance to the larynx, whereby edema appears in this region and presents laryngo-stenotic phenomena which resemble the clinical picture of laryngitis.

*Stomatitis aphthosa* usually heals in eight or ten days. When relapses

occur from renewed infection the disease may continue for several weeks, and the disturbances resulting therefrom, such as the insufficient ingestion of food, give rise to marked emaciation.

**Bednar's Aphthæ.**—Another local infection of the oral mucous membrane, due to cocci, is produced by *Bednar's aphthæ*, which are most prevalent in nurslings.

These form symmetrically round or elongated losses of substance upon the palate at the point where the mucous membrane covers the hamulus pterygoideus. This constant seat and the symmetric appearance of the ulcers appear to be favored by peculiar anatomic and physiologic conditions which dominate this area of the oral cavity. The mucous membrane of the mouth is exposed to a constant muscle-tugging produced by the movements of the jaws and also by the strong compression of the tongue against the hard palate.

Immediately following birth the hyperemia, previously described, appears upon the oral mucosa, which usually becomes irritated in consequence of the aforesaid influence exerted upon the palate. Constant pressure loosens the epithelium and causes desquamation, whereby lesions and ulcers appear. These excoriations form a suitable culture media for the cocci which find their way into the oral cavity, and a purulent infiltration of the mucous membrane arises in these areas. This condition may be induced by staphylococci as well as by streptococci, and, according to some authors, even by gonococci.

Corresponding to this conception of the causative factors Bednar's aphthæ commonly develop during the first week of life, and often twenty-four hours after birth; only exceptionally are they observed in older infants.

These aphthæ are usually superficial. By destruction of tissue they may invade deeply and form ulcers with sharp borders and a gray-coated base. As a rule, however, the ulceration remains shallow, but may extend from its inner border and thereby form a raw surface which distributes itself over the soft palate. By this process both aphthæ may appear to be connected by the purulently infiltrated mucosa.

Microscopic investigation shows that Bednar's aphthæ consist of necrotic elements, epithelium, pus-corpuscles, lymphoid cells, numerous cocci in the necrotic tissue, which permeate into the dilated vessels of the mucous membrane.

In a few days the gray mass which covers the base of the ulcer desquamates, the loss of substance is offset by the formation of granulations, and, with the formation of the epithelial covering, healing is complete.

This growth of epithelium begins at the border and healing takes place without cicatrization.

If the affection has distributed itself over wide areas of the mucous membrane the disease is prolonged and recovery by the formation of new epithelium is delayed. This is especially the case when there has been deep invasion of the tissues.

A common *complication* of Bednar's aphthæ is the *oidium albicans*, which greatly retards recovery.

In well-nourished children, and with proper care, Bednar's aphthæ heal rapidly. In debilitated children who are artificially nourished the affection may be very tenacious. The prognosis is generally much more favorable in children fed with breast-milk than in those who are otherwise nourished.

### III. BACTERIA OF DECOMPOSITION

The development of *putrefactive bacteria* in the decomposed remains of food which accumulate upon the gingiva, upon the teeth and between them, gives rise to an *especially characteristic inflammation of the tissues* which changes them into a pulpy mass and terminates in necrosis. In this affection Frühwald discovered a bacillus which grows upon gelatin and disseminates an odor of decomposition. In my opinion this process is the effect of various forms of putrefactive bacteria.

**Stomatitis ulcerosa.**—The regularity with which the process leads to necrosis of the gum, its tendency to invade deeply, its transmission to neighboring areas of the oral mucous membrane, the circumstance that *ulcerative stomatitis* occurs only in children who already have teeth and most frequently between the fourth and tenth years, and that the affection is rare from the first to the third year when greater attention is bestowed on the care of the mouth, are indirect proofs of the action of putrefactive bacteria as causal factors. As a further support for this view, the disease in question is most frequently noted in children with carious teeth or in the course of, or during convalescence from, severe infectious disease.

It is supposed that decomposed particles of food which are retained on the gums and between the teeth produce irritation and inflammation in these areas and that this condition occurs most readily and rapidly in anemic children. These swollen and inflamed regions are frequently squeezed and injured in chewing so that the putrefactive bacteria which are present have ready access to the tissue and cause necrobiosis. Present experience indicates that several varieties of the bacteria of decomposition are present simultaneously.

*Symptoms.*—The *symptoms of ulcerative stomatitis* are as follows:

The gum, either in a circumscribed area or over the entire jaw—in well-marked cases upon both jaws—becomes dark red and loosened. The lower margin is swollen and bleeds readily upon contact; simultaneously a putrid odor is diffused from the mouth; salivation is increased and other areas of the oral mucosa are also reddened.

As the disease progresses the external free border of the inflamed gum alters its color and becomes a yellowish mass; if this is brushed with cotton the mucous membrane beneath appears livid, bleeds readily and has an excoriated appearance.

Necrosis of the gum thus begun usually distributes itself downward and



may change the entire area into a necrosed mass. The space between the teeth and the posterior surface may be implicated by this decay, so that in severe affections the teeth are entirely denuded of gum.

During this process usually there is enlargement of the submaxillary glands.

When the necrotic process of the gum is fully developed, those portions of the oral mucosa which come in contact with the necrosed parts when the jaws are closed (lower and upper lips, cheeks, margin of the tongue) are implicated in a like manner. These areas form inflammatory foci which, in their course, become grayish yellow, and, after desquamation of the epithelium, are changed into a necrosed mass which exudes an exceedingly fetid odor. After these masses are expelled there are irregular losses of substance with a grayish yellow base surrounded by a swollen mucous membrane with serous infiltration.

The malady usually *terminates* in recovery. The offensive odor of the mouth disappears with the desquamation of the necrosed tissue, salivation decreases and the loss of substance is gradually replaced by granulation.

*Noma* may result from the deep invasion of the process in the cheek; if the periosteum is implicated there may be partial necrosis of the jaw.

The *course* of the disease is either acute or chronic.

When the process lasts for a long time the nutrition of the body suffers and death may occur from general sepsis.

The *diagnosis* is dependent upon a careful microscopic examination of the products of the disease and upon the sequence and nature of the symptoms. By a strict utilization of the points of support which have been mentioned a confusion of this affection with any other disease of the mouth is hardly possible.

In well-nourished children the *prognosis* is favorable. In debilitated patients and after a prolongation of the disease the termination may be fatal.

## PROPHYLAXIS AND TREATMENT

To *prevent* the development of the above-mentioned infections of the oral mucosa in children it is necessary for the physician to give attention to the cleansing and disinfection of the oral cavity.

*Prophylactic antisepsis* is governed by the age of the child, the condition of the mucous membrane, and the nature of the threatening infection.

Above all, *routine washings* and *improper treatment* of the oral mucous membrane, so commonly practised by uneducated nurses, must be prevented. All articles by which the child is fed (spoons, glasses, rubber nipples, etc.) are to be *sterilized* before use. In breast-fed children the nipple should be bathed before and after nursing with a one per cent. boracic acid solution.

*Infection by saprophytes* may be prevented by cleansing the oral mucosa of the new-born once or twice daily with sterilized water. This may be best accomplished with a cotton tampon.

When older children retain food in the mouth because of the large quantity ingested, or as the result of insufficient mastication and improper deglutition, a careful washing of the mouth after each meal with distilled water to which some aqua menthæ or lemon juice has been added is serviceable.

For the prevention of *catarrhal stomatitis* or *chemical decomposition*, due to *saprophytes*, the following is a useful antiseptic mouth wash:

R/.

Acid. bor.....	3.0
Aq. font. dest.....	200.0
Tinct. myrrh.....	2.0

M. Sig.: Rinse the mouth three times daily.

The teeth must be cleansed thoroughly to keep them in good condition and to prevent fermentative processes and decomposition. For this purpose the following tooth powder may be employed:

R/.

Magnes. carbon.....	5.0
Cret. alb. praep.	
Natr. salicyl.....	15.0
Ol. menth. pip.....	gtt. vi

M. Sig.: Tooth powder.

When a fetid odor emanates from the mouth in consequence of fermentation and putrefaction the following solution is of value for cleansing the oral mucous membrane:

R/.

Natr. salicyl.	
Natr. phosphor.....	2.0
Aq. font. dest.....	300.0
Ol. menth. pip.....	gtts. v

M. Sig.: Mouth wash.

*Infection* of the oral mucosa by *pathogenic bacteria* requires special therapeutic treatment in addition to the foregoing methods.

In infection by the *oidium albicans* all food-stuffs which appear to favor the development of this parasite, such as sugars and syrups, should be avoided.

As this bacterium is present in the air, thorough ventilation of the bedroom is to be urgently advised, particularly in institutions.

The mechanical removal of the deposits of *oidium albicans* without injury to the epithelial layer is indicated. The mouth should be washed with a cotton tampon which has been dipped in a 2 per cent. solution of boracic acid or sodium borate. A fresh tampon should be employed for each portion of the oral cavity, and every fold and niche must be carefully cleansed. A 5 to 10 per cent. solution of sodium borate or a 3 per cent. solution of sodium

benzoate is very serviceable for this purpose. Some authors advise solutions of potassium permanganate, resorcin, and even corrosive sublimate. Careless employment of these remedies is dangerous and I therefore do not advise them.

Prior to each of these manipulations in the mouth the nurse must carefully disinfect her hands with soap.

When *gonococci* are present in the oral mucosa I employ

R/.

Alum. crud..... 0.5

Acid. carbol..... 0.2

Aq. font. dest.....200.0

The mouth should be cleansed with cotton tampons, as in infection by *oidium albicans*.

In *staphylococcus* and *streptococcus* infection tannin may be used as a dusting powder, or washes of a 2 per cent. solution of tannin; in severe cases iodoform or sozoidol may be employed.

As this infectious disease is very painful lukewarm food only is advised.

To prevent deglutition of the cocci, the oral cavity should be cleansed before each meal with a 2 per cent. boracic acid solution.

In this infection the *avoidance of certain articles of diet*, such as pap, infant foods and milk sugar, is essential. In severe processes the mother's milk is the best food; where this cannot be obtained I employ a mixture of milk and whey, which I call infant milk.

Ventilation of the room and scrupulous cleanliness (removal of dirty bath water, soiled sponges, etc.) are of the utmost importance for the prevention of renewed infection.

In *aphthous stomatitis* the same general antiseptic rules apply, and, in addition, the mouth should be cleansed several times daily with antiseptic fluids (boracic acid, tannic acid).

Because of the pain only cool fluid nourishment should be administered, and the mouth should be frequently rinsed with cold water.

The following mixture may also be employed:

R/.

Kal. chlorat..... 3.0

Aq. font. dest.....200.0

Tinct. myrrh..... 3.0

When pain is intense and there is confluence of the aphthæ I advise the employment of corrosive sublimate solution:

R/.

Hydrarg. chlor. corros..... 0.1

Aq. font. dest.....100.0



Some physicians employ potassium permanganate, tannoglycerin 1 to 30, or cocain, similar to the following:

R/.	
Natr. salicyl.....	0.1
Cocain. hydrochlor.....	0.2
Aq. font. dest.....	10.0

M. Sig: Cautiously applied in the mouth.

*Bednar's aphthæ* are to be treated likewise. The deep ulcerations should be dusted with iodoform or sozoioidol.

In *ulcerative stomatitis* the following measures are to be employed: Some cotton is swabbed around the tooth and the necrosed areas of the gum are thus removed, the bleeding borders being treated with a 2 per cent. solution of carbolic acid. If necrosis has not yet developed, frequent washing with the following solution is of service:

R/.	
Kal. chlor.....	3.0
Tinct. ratanh.....	5.0
Aq. font. dest.....	200.0

When the necrosis permeates deeply, careful dusting with iodoform is to be employed to prevent infection of the neighboring areas, and if the mucous membrane of the cheek is invaded the Paquelin cautery must be applied to prevent the development of noma.

[Frequent contact of the chlorate of potassium or of sodium is advisable in most of the forms of stomatitis, mainly the ulcerous. At the age of 1 or 2 years one gram—15 grains daily is tolerated without injury. That may be dissolved in 150 c. c.—5 ounces of distilled water, of which  $\frac{1}{2}$  teaspoonful may be swallowed every 15 minutes while the baby is awake. Thus there is a constant local contact, beside the general effect.—EDITOR.]

# ACUTE DIGESTIVE DISTURBANCES OF INFANCY

By TH. ESCHERICH, GRAZ

## HISTORY

Because of their frequency and danger the digestive disturbances of infancy have long occupied the attention of physicians, even before the establishment of pediatrics as a substantive science. In the works of Aretaeus, Abercrombie, van Swieten, and others, detailed descriptions have been devoted to these diseases. A more intimate understanding of their peculiarities, however, developed simultaneously with the special investigation of diseases of infancy, and one of the principal factors which brought pediatrics into prominence was the devastation which gastro-intestinal affections had wrought among infants, especially in foundling institutions.

The first fundamental results in this science were achieved at the beginning of the nineteenth century, in the illustrious period of French medicine to which we are indebted for the foundation of modern diagnosis. Under the mighty influence of Broussais's idea the first rational treatises of the pathologico-anatomical conceptions and findings were published by Denis (1826) and Billard (1828), and were soon followed by those of Louis (1829), Valleix (1838), Barrier (1841), Legendre (1846), Hervieux, and others. The active interest which was devoted to this subject by the whole science of medicine is evinced by the participation of Trousseau and Cruveilhier in the elaboration of many of the important problems of pediatrics.

German pediatrics, which was still involved in nature philosophy, and was directed into wrong paths by the incorrect teachings of Jäger regarding gastromalacia, has as yet nothing equally brilliant to offset this. Only during the 50s of that century did new and substantive methods enter into the realm of gastro-intestinal affections, and these were introduced by the Austrian school in the foundling asylums of Vienna and Prague. To this period belong the labors of Bednar (1850), v. Ritter, Steiner, Lambl, and Mayr, and the culmination was reached in the comprehensive description of Widerhofer (1880) in Gerhardt's Handbook of Diseases of Children. The pathologic types which arise in infancy, the semeiotic and the pathologico-anatomical changes, in so far as they could be recognized macroscopically, were so exhaustively described that up to the present time nothing essentially new has been added. On the other hand, our knowledge of the etiology and pathogenesis of these affections, which alone would permit an insight of their nature, was still exceed-

ingly scant, and no means could be discovered to illumine this obscure realm other than by pure clinical observation.

Meanwhile in Germany, a method rapidly developed from modest beginnings—the study of physiology and artificial nutrition and the problems connected therewith—which gave promise of a more hopeful result. A brief description will be devoted to this last aspect, which still holds our attention.

First, we will consider the investigations of Biedert regarding the difference between breast milk and cow's milk, and the conclusions he deduced therefrom which unfolded the scientific theories of artificial nutrition. The fundamental idea in this was the injuriousness of casein, which, according to the knowledge then extant, represented the principal difference of cow's milk in comparison with human milk. It therefore followed that this was the cause of the surprisingly high morbidity and mortality of artificially fed infants—a view which was confirmed early in the fourth decade of the last century by the investigations of Rau, Lichtenstedt, and others.

Bacteriology, which was founded by Pasteur and Koch, revealed the presence of numerous small living organisms in cow's milk, which established a further important difference. Similarly as in surgery, here also a practical achievement, the invention of the Soxhlet apparatus, smoothed the way for scientific theories. At the Congress of Natural Philosophers at Wiesbaden in 1887 the view was first promulgated that bacteria are the most frequent and most dangerous generators of digestive disturbances. To-day this view is general, not only in regard to the digestive system but in every region of pathology, and only as to whether bacteria produce disease by direct invasion of the intestinal wall, or indirectly by decomposition of the deleterious residue of food, are there differences of opinion.

In addition to the above-mentioned variations in the results of artificial and natural nourishment, we have the following objective facts upon which the bacteriologic hypothesis is based: 1. The increase in the frequency and severity of diseases during the hot season, especially in artificially fed children—first noted by American authors (Dewees, 1833). According to Baginsky (1875) the only explanation of this increase is that under the influence of the higher temperature, which favors bacteriologic decomposition, deleterious agents are propagated which produce disease in the infantile organism. 2. The occurrence of epidemic cholera infantum, as was first noted in breast-fed children in foundling asylums, and later in those artificially nourished in the infant department of the Berlin and Graz Children's Clinic. 3. The character of the evacuations, which, in the healthy child, contain a uniform flora of few varieties, while in intestinal diseases additional bacteria, particularly those of a pathogenic nature, are present which are foreign to the normal feces,—therefore, conditions which favor a bacterial infection.

A third view, founded by the Breslau school, refers these disturbances to faulty nutrition, etc., due to the intermediary metabolism. This theory is invoked principally in explanation of chronic digestive disturbances which are not to be considered here, and it may therefore be disregarded.



Parallel with these investigations, and in great part stimulated by them, there was a renewed and more profound study of the physiology of the digestive tract with special reference to problems of artificial nutrition. The ingenious explanations of Boër, Jörg, and Pollitzer, and the anatomical investigations of Fleischmann may be regarded as introductory. The analytic and experimental researches began with the labors of Wegscheider (1875), Zweifel, Korowin, Baginsky, Gundobin, and Pfaundler, and demonstrated conclusively the functional inferiority of the digestive apparatus, which to that time had been only surmised. Of special importance also was the study of the intestinal bacteria in the nursling, which was begun in my monograph, published in 1886, but which has not yet been concluded notwithstanding the excellent work of Tissier. Finally, we must mention the most recent experiments in energy and metabolism which illuminate a new and hitherto entirely neglected portion of the nutritive process.

Thus there has been a complete change in the direction of investigation. Whereas formerly the direct discovery of the nature of the disease was attempted by careful clinical observation and by the study of pathologico-anatomical changes, especially in affections of breast-fed children, which were quite properly regarded as the purest types, at the present time we attach special importance to investigation of the physiologic conditions. From this groundwork the realm of pathology is entered, in that the reaction of the intestine to definite irritation has been studied, and the degree of the irritation accurately determined. Artificial nutrition, which is in itself no more than an experiment on the normal nursling, is incomparably better in these physiopathologic experiments than breast feeding wherein a number of unknown and incalculable factors must be considered. Besides, it is the kind of nutrition which is prevalent to-day and also, as already stated, the most frequent cause of disease and death from digestive disturbance.

It is understood, therefore, that the study of the pathogenesis has developed conjointly with the problem of artificial nutrition and especially with the observation of artificially nourished infants. Although much labor has been expended on this painstaking method, only slight and disconnected results have been forthcoming; nevertheless we may hope that eventually, by a synthesis of these results, we shall gain a positive experimental fundament upon which a pathogenesis may be erected. To-day we are far removed from such a goal. True, bacteriology has revealed a number of new and important facts and has furnished explanations for other already familiar data, and in therapeutics and prophylaxis has produced unquestioned results; but so long as the connecting link—the knowledge of the toxins and their effect upon the infantile organism—is lacking it must remain only a likely hypothesis. Even as such, however, bacteriology has greatly influenced the investigation of this subject, which has been so long neglected, and the major part of our knowledge of the pathogenesis of digestive disturbances and almost everything we have learned of the etiology of acute gastro-intestinal affections were acquired along this path. Notwithstanding the new view-

points which have already arisen in the present century it may for a long time govern the trend of investigation. It seems to me well to devote a chapter to bacteriology in this article. In the following I shall attempt briefly to explain the general conceptions and conclusions that have been deduced by recent bacteriologic investigation in the pathogenesis and treatment of acute digestive disturbances of infancy, and in this connection reference must necessarily be made to the earlier researches.

## PHYSIOLOGICAL ASPECTS

Gastro-intestinal disease always produces a disturbance in the physiologic processes; therefore a knowledge of the functional peculiarities of the infantile organism is absolutely necessary for an understanding of the pathogenesis. In no organ of the new-born and infant is there such a disproportion between functional arrest and requirement as in the digestive tract. I shall not enter upon a dissertation of the much discussed deficiency in ferments and the muscular weakness of the infantile intestine, but must emphasize one point which in my opinion is at present considered much too little. This is the shortness of the complete intestinal canal (about 3.5 meters, against 8 to 10 meters in the adult), which is not compensated for by the great relative length and capacity which are so frequently emphasized. The entire length of the bowels of the newborn would extend in the adult only to the lower third of the small intestine, and hence it is apparent that, reckoned upon a similar length of the intestinal passage the power of absorption is more favorable in the nursling. Nevertheless the shorter length of the entire intestine of the newborn and infant together with the smaller number and size of the glands permits no more digestive capability that is always required for the assimilation of breast milk which the child consumes. Overfeeding, especially by artificial means, will exceed this physiologic limit and may therefore have a deleterious effect. It is true, a certain excess of breast milk, as well as of cow's milk properly prepared and administered, is ingested by nurslings without causing disturbance, but the tolerance as opposed to the deviation from the physiologic condition, the assimilative capacity for a too profuse or foreign food, although it shows individual variation, is nevertheless strictly limited, and becomes more circumscribed the weaker and younger the child. This absorptive quality increases markedly during the second half-year, and in the further development the digestive organs acquire that wonderful faculty of compensation which makes it possible for man to satisfy his nutritive requirements under the most varied conditions and zones.

Furthermore, there is the special irritability and vulnerability of the infantile mucous membrane to mechanical and chemical stimulation. The physiology of the newborn abounds in examples of traumatic effect produced by contact with the media of extrauterine life; then how much greater the injury from filling of the intestinal canal with food, gases, bacteria, etc. In

the mucous membranes which are exposed to contact with the external wall we observe a process which we designate as *habit or hardening*, whereby irritation, even feeble or slight, at first produces a severe reaction but eventually shows little or no effect. The delicacy of the anatomical relations, the vascular richness of the mucous membrane (Gundobin), the greater amount of water in the tissues, the relative preponderance of the nervous elements, have all been invoked in explanation without striking the kernel of the matter. Not only is the irritative stimulus decidedly lessened, but also the reaction is uncommonly turbulent, for there is an early loss of epithelium, and inflammatory processes develop in the tissues which may distribute rapidly over large areas of the intestine and retrogress very slowly even after the irritative cause has been removed. This peculiarity has an important bearing on the origin and course of the digestive disturbances of infancy, and is all the more significant because, in all of the tissues of the infantile digestive tract, there is a special susceptibility and predisposition to bacteria and their products of metabolism.

Proof for this is furnished by the pathology of the newborn, which shows such a severity, frequency, and multiplicity of septic diseases as is seen at no other period of life. To exemplify the special predisposition of the digestive tract I may call attention to the oral mucosa, which in young children is so commonly the seat of mycotic disease, but from the third year of life is only exceptionally attacked, notwithstanding the increased opportunity for infection. Here, similarly as was demonstrated by Krieger for the respiratory system, we find a *local immunization of the mucous membrane* of the digestive tract,—a hardening process proceeding downward from above. Later we will consider the toxic action exerted upon the nursling by even slightly decomposed cow's milk which would not be experienced in an older child.

In positive contrast to these views is the fact that the nursling possesses a normal or profuse *intestinal flora* which apparently exerts no deleterious action notwithstanding the fermentative and even pathologic property of the bacteria. An understanding of this condition is impossible in the unsatisfactory state of our knowledge, but the uniformity of the varieties of intestinal flora, as well as a study of their biologic properties proves that we are dealing with an autochthonous vegetation, suited to the special conditions and to the individual, which has no analogy with the bacteria found in cow's milk or in the decomposed material. On the contrary, we must assume that the intestinal bacteria play a useful rôle in the process of normal digestion, although by what means is not fully understood. This is obvious from the fact that a change of vegetation and the introduction of abnormal fermentative processes are sufficient to disturb the health of the child and the course of its digestion. Such is the origin of the fermentative dyspepsias which are peculiar to early infancy. Their frequency is explained when we consider that milk, the exclusive food of the child, is especially liable to bacterial change and fermentative processes because of its composition and percentage



of water; furthermore, in the nursing conditions obtain which decidedly favor the entrance and increase of foreign organisms in the intestinal canal. Even with a food poor in bacteria, the abundance of the microorganisms in the oral cavity will lead to infection of the stomach. Here their brief residence, and the admixture of the scant amount of hydrochloric acid already present with the salts and albumin of cow's milk, hinder destruction so that even normally a large or smaller amount of chyle which contains bacteria passes into the duodenum. Strictly speaking, the intestine appears, as is obvious from recent investigations, to have a selective bactericidal property which is directed only to foreign bacteria, not to those which it requires. It is quite likely, although not yet proven, that this protective measure is more feeble in the nursing. This is indicated by the care which nature has taken to prevent the entrance of germs into the natural food and to avoid processes of decay in the intestinal tract.

Digestive disturbances have an especially deleterious effect upon the infantile organism. The milder forms produce fluctuation and arrest of the growth of the body, the more severe disturbances lead to a rapid decrease in weight and diminish the resistance of the body to all deleterious agents; hence they give rise to manifold secondary diseases. In addition, the course of these diseases in infancy is relatively brief and pernicious, whereas in the adult the affections of this tract are mostly secondary, and are milder and more prolonged.

## ETIOLOGY

The foregoing observations must be borne in mind when we consider the *causes of digestive disturbance*. These may be separated into *four groups*: The first comprises derangements in the anatomical and functional development of the digestive tract. The second group is formed by injurious alimentation, which may be referred to quantitative or qualitative errors of nourishment. The most important among these are the changes due to bacterial decomposition which the milk undergoes within and outside the intestinal canal. The third group includes the actual infectious processes which owe their origin to an invasion of the intestinal tract by pathogenic bacteria, irrespective of the nature of the food. In the fourth group are classed those causes which have not already been mentioned, such as protozoal, mechanical, chemical, thermic, nervous irritation, etc., and especially physiologic conditions. The essential and most characteristic feature of these causes of disease, which, with the exception of the first group, also appertain to adults, is this, that even in their milder grades of intensity they act upon the susceptible infantile organism with such severity and frequency that more than half of all diseases, and a third of all deaths, in this period of life are due to them. These children are made ill with food which would be a most harmless sick diet for an adult or even for a child of two years, and it is exceedingly difficult to determine—in the manner of its acquisition, preservation,

and administration—the nature of the change which is the cause of disease in the individual case. This *microscopic smallness of the causes*, if I may use such a term, is far beneath the stimulus which would be operative in an older organism, and is also the reason why this particular realm of pathogenesis has been entered so late and only with resource to newer methods of investigation.

**Group I.**—The first group, *the anatomical and functional insufficiency of the digestive apparatus*, shall be only briefly considered. The cause is obvious, and is also the result of undigested remains of food in the feces, commonly known as dyspepsia, so-called by Bamberger. If the activity of the digestive apparatus is defective or insufficient for physiologic requirements, we speak of an *organic dyspepsia*. If, upon the introduction of an amount of food greater than the individual property of assimilation, there is a disproportion between requirement and activity, which has already been considered, we are dealing with an *alimentary dyspepsia*. This forms the transition to the second group of *alimentary deleterious causes*.

**Group II.**—The food, as well as the excessive introduction of fluid which is not absorbed in the intestinal canal, produces under some circumstances, as in nutrition with cow's milk, an abnormal weight and damage to the muscular elements by a clumpy coagulation in the susceptible digestive organs or by direct mechanical irritation. This condition is much aggravated by the undigested residue of food, which produces a change in the chemical composition of the intestinal contents and a disturbance of the normal vegetative relations. The rapid and complete absorption of the mother's milk is undoubtedly one of the most essential requirements for the maintenance and uniformity of the normal intestinal flora. A change in these conditions, even such as occurs in nutrition with carefully sterilized cow's milk, gives origin to other vegetative and nutritive conditions of bacteria and also to a change in the secretion, the reaction of the intestinal contents, and the peristalsis, which cannot be calculated. As a matter of fact, in the feces even of children nourished with cow's milk and who show no symptoms of disease, a *bacterioscopic picture* is presented which deviates from the norm, in that we see more or less facultative, saprophytic bacteria in addition to those which are commonly encountered. This proves that the bactericidal faculty of the intestinal canal is weakened by these conditions and its resistance to the colonization and increase of foreign microorganisms is much diminished. In this sense Biedert may quite properly refer to "deleterious remains of food," or, more correctly, to "deleterious agents of the food," because every deviation from physiologic conditions creates a breach in the thin boundary line which here separates the normal from the pathologic.

However, these deviations have more of a predisposing influence so long as they are not combined with definite bacterial processes, and are only exceptionally of substantive pathogenic importance.

The assumption of a special damage from bacterial decomposition of the milk or of the intestinal contents is based upon the statistical fact, already

mentioned, of the much greater morbidity and mortality of artificially nourished children in general, and especially during the hot season. We might mention a third factor, the social life, to which all writers upon the mortality of nurslings refer. But this influence is operative only in the sense that deficient care in nursing and cleanliness, which is common among the poor, tends to a greater contamination and decomposition of cow's milk because of its much more complicated preservation and preparation, especially during the summer.

Prausnitz demonstrated in his Graz statistics that the wealthy classes are not represented in the official lists of infant mortality in the gastric and intestinal affections, therefore we may set aside the objection propounded by Flügge that the introduction of the Soxhlet apparatus has caused no decided decrease of gastrointestinal affections in the mortality of infancy. Those classes of the population for whom the Soxhlet apparatus is available are largely or altogether excluded from the statistics. Besides, the personal experience, no matter how limited, of every pediatricist, proves that natural germ-free food is far superior to artificial nourishment with its deleterious effects of heat and uncleanness, or to the employment of sterilization. I shall not therefore exemplify these statements but will now give more detailed consideration to the changes which bring about decomposition.

The view that the germs which inhabit cow's milk are the cause of digestive disturbance was first promulgated by Hessling (1866) and Meissner (1878), although Bednar (1850) had already quite correctly associated the diarrheic discharges of nurslings with fermentating milk. But not until the introduction of Koch's method was it possible to estimate the number and nature of these germs. In collaboration with Dr. Knopf (1889) I have calculated that in milk obtained by cleanly methods the number of germs, a few hours after milking, averages one-half to one million per cubic centimeter.

The so-called spontaneous lactic acid fermentation of milk which ensues immediately after milking is not, as Pasteur presumed, a uniform process, but is due to a number of schizomycetes, among which we may distinguish saccharolytic, proteolytic, and perhaps also pathogenic bacteria. The saccharolytes are the richest and most numerous group and are represented by several varieties. They decompose the milk sugar and produce organic acids, thereby inhibiting the increase of the proteolytes and finally of all bacteria with the exception of some few acidophilic varieties.

The nature and rapidity of these processes is dependent upon the microorganism, upon the time which has elapsed since milking, and upon the temperature at which the milk is kept. In another article (*Wiener med. Presse*, 1889) I have stated that the height of the temperature is of influence not only upon the number of bacteria but also upon the nature of decomposition, in that milk kept at a high temperature (35° C.—95° F.) propagates microorganisms—probably toxic bacteria and products of metabolism (succinic acid, alcohol)—which do not develop in milk kept at a lower tem-



perature. Young dogs were made ill with milk kept at the body temperature and one of them succumbed with choleraic symptoms, while milk from the same source, kept at a lower temperature, was administered without effect. These observations, together with those of Scholl and Schierbeck regarding the greater virulence of fermentative bacteria in summer, explain the increased toxicity of milk during the hot season. Accurate chemical investigations in regard to the formation of toxic substances are unfortunately not at hand. Vaughan reports the isolation of a toxin-forming colilike bacillus, and a toxin produced by it which he designates tyrotoxin. The action could only be observed in young mammals, which perhaps have the same susceptibility as the child. Thus far we can only presume their presence when the milk shows evidence of advanced bacterial decomposition. As a matter of fact E. Plant has demonstrated that milk which has been kept in the homes of children who have been attacked by summer diarrhea has a very excessive acidity.

Milk that has been incompletely sterilized undergoes a different change, whereby the asporogenic varieties of saccharolytes are mostly or entirely destroyed and the resistant proteolytic and anaërobic bacteria as well as others which have accidentally gained entrance to this media multiply unhindered. Although the pathogenic importance of the proteolytes and anaërobic bacteria has been greatly exaggerated, nevertheless toxic substances may develop even after sterilization if the milk is kept a long time (Carstens). In how far the toxins which are formed in fresh and in incompletely sterilized milk are influenced or destroyed by heat is unknown, but clinical experience favors the view that milk which is much decomposed cannot be made altogether harmless even by prolonged sterilization (Marfan).

All of these processes which run their course in the milk outside of the body I designate *ectogenous milk decomposition*, in contrast with *bacterial decomposition* which the milk or its individual constituents undergo in the intestinal tract after ingestion. As is well known, a bacterial decomposition of the milk sugar occurs normally from autochthonous bacterial vegetation, especially of the bacterium *lactis aërogenes*. This process, by maintaining a moderately acid reaction and forming gases which fill the intestinal coils, may serve a useful purpose. The coli-, anaërobic, and acidophilic vegetations which predominate in the large intestine of the breast-fed nursing do not attack the residual food. According to Biedert, in children nourished with cow's milk the unabsorbed casein produces a normal process of decomposition which can be distinguished by the alkaline reaction and by the odor. In view of the frequent confirmation of the lesser tendency of casein to decomposition further proof must be forthcoming to determine whether, in this not unusual process, there is a change in the increased intestinal secretion. Unfortunately, even in the digestion of cow's milk, we lack a knowledge of the usual method of bacterial decomposition.

The pathologic deviation may occur as an excess of this normal process, whereby the abnormal amount of acid produces irritation of the intestinal

wall. Baginsky has assumed that the bacterium perishes in its own acid and that other bacteria do not enter until it has succumbed.

I believe that an excess occurs especially from the distribution of the bacteria in the intestinal canal, in that this process, which is usually limited to the small intestine, invades the entire length of the intestinal canal when carbohydrates are introduced in large amount or are slowly absorbed. Then, in addition to the symptoms of acid diarrhea and remains of sugar or starch, we find large numbers of the bacterium lactis in the feces. Booker has also observed the frequent appearance of the bacterium in the diarrheic discharges, but he regards this only as the result of the more rapid evacuation of the intestinal contents.

Much more frequently we observe that foreign vegetation ejects the normal and occupies its place. This occurs most simply by a direct continuation of the ectogenous fermentation, as after the ingestion of decomposed animal milk. In other cases these germs are accidentally introduced with the food, with the saliva, or otherwise, and find opportunity for accumulation (*chyme infection*) in their passage through the intestine. However, such an increase cannot be considered unless the normal process of digestion has previously been destroyed and the bactericidal power of the intestine weakened.

The nature of this decomposition which takes place in the intestinal canal follows the same laws as those which are operative outside the body: So long as there are soluble carbohydrates (milk sugar) in the intestine the decomposition is saccharolytic, and at the same time the resulting acid reaction prevents the development of putrefactive processes (Hirschler, Seelig). Putrefactive changes of this nature are possible only in the lower parts of the intestine, where the sugar has already disappeared by resorption. This eventually leads to the formation of offensive products and an alkaline reaction, and I have therefore designated the process *alkaline fermentation* (1889). Here, as already mentioned, the albuminoid secretions of the intestine, which are readily decomposed, are active and very likely preponderate.

Furthermore, according to the latest investigations of Tissier, anaërobic bacteria, which find suitable conditions for their development in the intestinal tract, may give rise to disease more frequently than was previously assumed. I can present only a single observation in proof of this,—a case of very acid effervescing feces with the odor of butyric acid, in which the anaërobic immotile bacillus butyricus (Botkin) could be demonstrated in large numbers. Unfortunately, notwithstanding the extensive bacteriologic examinations of the dejecta, very few tangible facts in this direction have been obtained.

This lack of result is especially true as regards the generators and the products of acid fermentation. In this direction there are no definite reports with the exception of the demonstration of butyric acid by Ludwig (reported by Widerhofer), and Schlossmann's report regarding the probable fate of starch in the intestine of young children. On the contrary, the attention of investigators was devoted to albumin decomposition, which Baginsky

regarded as the principal source of intestinal intoxication. In his report of 1891 he infers that saprogenic germs are primarily capable of forming toxic peptone-like bodies from the albumin present in the food until the production of a decided quantity of ammonia concludes the process. The totality of the substances formed and their entrance into the lymph tracts and circulation are the cause of the severe clinical symptoms. Up to now only the *proteus vulgaris* has been isolated as a generator of decomposition (Booker), while the *bacillus putrificus coli* (Bienstock) has been found in meconium but not in the feces.

In my opinion Baginsky has exaggerated the importance of the processes of decomposition in pathogenesis. Casein, the principal proteid of milk, tends but little to decomposition and when such a process occurs in the intestine of the nursling I rather agree with Czerny that it is due to the readily decomposed proteids of the intestinal secretion. Furthermore, the most severe toxic symptoms such as are seen in acute cholera infantum, run their course with a decidedly acid reaction of the intestinal contents and of the feces. Further research must explain this apparent contradiction. The toxins arising in milk and in the intestinal tract need not, as was for some time supposed, owe their origin to destruction of albumin bodies, but may be formed synthetically, as Blumenthal has shown, by saccharolytic bacteria. I would also suggest that although all of these processes depend upon the presence of bacteria, with improvement in our technic we may discover microorganisms which are common to all processes of fermentation and decomposition.

I have proposed the term *chyme infection* for the endogenous processes of decomposition, i. e., those occurring in the intestinal canal, to denote that this change first occurs in the intestinal contents at its expense and with a change in its chemical composition. The intestinal wall is only indirectly affected by irritating products thus formed; the remaining organism by the absorption of contingent toxic material.

**Group III.**—The *third group, true intestinal infection, disease of the intestinal wall itself*, is in contrast to this in that the surface becomes the site of an infectious catarrh likewise as in the bronchial mucous membrane, or where there is a direct invasion of the mucosa by specific tissue parasites. This is analogous to the localized intestinal infections of adults: enteric fever, Asiatic cholera, and dysentery. A typical example of infectious catarrh is furnished by staphylococcus enteritis of over-fed breast children, explicitly described by Moro. In these cases the pathogenic agents are probably identical with the staphylococci found in stagnant breast milk. Streptococcus enteritis—pyocyanous diarrhea—also belongs to this group. In fact the majority of microorganisms pathogenic in man, especially those endowed with septic or pyogenic qualities, are capable of producing infectious catarrhs or inflammations when they accumulate in the intestinal tract.

The conception of these cases is similar but is not entirely parallel to Epstein's view, according to which gastro-enteritis, no matter under what



name all of the inflammatory intestinal diseases have been included, is only one of the many expressions of a septic general infection. We both admit that the ordinary causes of sepsis are capable of producing this disease, but Epstein believes that the intestinal disorder represents only a partial phenomenon of septicemia, no matter how acquired, while I would exact, for the assumption of a primary intestinal infection, the proof that the pathologic process has originated in the intestinal canal, and eventually also the demonstration of the pathogenic cause in the diarrheic discharges. Corresponding to the nature of these bacteria there is usually a deep lesion of the tissue and in some instances even the development of sepsis from the intestine. This process, however, apart from the agonal invasion, is rarely observed and has thus far been demonstrated conclusively only in streptococcus enteritis.

The much more common form, which has become familiar from the excellent descriptions of Fischl, shows an inverse type wherein the diarrhea occurs in the course and as a partial phenomenon of sepsis, which usually originates by inhalation. However, I believe it to be more correct to include these under symptomatic diarrhea. In some few cases, it is true, it may be difficult or even impossible to decide whether we are dealing with a primary localization of sepsis in the intestine or with a symptomatic diarrhea following general sepsis. Mixed forms may also occur; for example, an ulcer in the mouth may lead to general infection, and simultaneously, by swallowing the pus, give rise to an infectious catarrh. This disputed question is of secondary importance, however, to the fact that here the disease is not caused by saphrophytic or fermentative bacteria but by specific organisms pathogenic to man which are independent of the nature of nutrition and climatic influences. This contrast is most noticeable in foundling asylums, where these diarrheas occur with special frequency and severity among the breast-fed children, and particularly in the cold season when there is usually overcrowding. This affection was for a long time supposed to be a peculiarity of such institutions, until the investigations of Lesage, Rossi, Finkelstein, and others showed that epidemic diarrhea was not uncommon even among artificially nourished children. These observations, together with the accurate histologic findings of Booker, led to a more accurate study of the schizomycetes present in the evacuations of such patients, and in some cases at least I found a characteristic fecal picture. The cases sometimes occur sporadically, sometimes in epidemics, or they may appear in groups and are then further distributed by contact infection. This epidemic distribution, which is common to older children, colitis infectiosa, as well as that described in the *Jahrbuch für Kinderheilkunde*, Bd. LII, belongs to bacillosis. In the former it appears that there are microorganisms belonging to the coli group which confer immunity and may be recognized by the serum reaction. In all essentials they coincide with the generator of dysentery, described by Celli, Shiga, and Kruse, but in my cases no connection with dysentery could be demonstrated. According to our present knowledge the bacilli found in

"bacillosis," stained by Gram's method and cultivated, I should include in the group of the acidophiles, which, as was shown by Finkelstein, also includes pathogenic microorganisms. I have formed no opinion of the nature of the pathologic process observed at that time.

The pathogenic faculties of the bacteria common to the feces of milk-fed children permit them under certain circumstances, like foreign infectious germs, to enter into the blood and the tissues.

**Group IV.**—The *fourth group* affords but secondary consideration in our discussion. There are few reports of the appearance of protozoa in nurslings—helminths may be entirely disregarded. In a limited number of affections I have observed the *Lambia intestinalis* (*megastoma entericum*) with amebæ, as well as a flagellate variety. In the last case only, which was published in a report of Cahen (*Deutsche med. Wochenschrift*, 1891) was I inclined to regard these as the cause of the symptoms. Epstein reports that during an epidemic among the nurslings in his foundling asylum a protozoön, designated *monocercomonas*, was found in great numbers in the evacuations.

The transmission of toxic products from the food to the milk has been proven in the human being as well as in the cow. In this way the organisms which produce fermentation may cause damage in the intestinal canal. Lesage reports that in the Paris Foundling Hospital, where once a week the wet-nurses have a fermentating cabbage as food, one-half of the children are attacked with diarrhea.

This also shows the peculiar sensitiveness of the infantile organism to substances which are borne by the adult without injury. The same result is observed in cows fed with brewer's grains and swill. However, Soxhlet quite correctly asserts that this manner of feeding may also have a directly deleterious influence upon the bacteria of the milk by unavoidable contamination of the latter with particles of feces.

That *thermic influences*, especially cold, may stimulate peristalsis and cause thin, fluid stools is well known to everyone who has studied metabolism in infants. Meinert is of the opinion also that excessive heat may give rise to choleraic symptoms. *Mechanical irritation* of the intestine is due to the presence of foreign bodies, undigested particles of food, and hard, fecal masses. The teaching of *dentition diarrhea* originates from a period when, in the absence of other recognizable cause, gastro-intestinal diseases were assigned to an external conspicuous process of development—dentition. The so-called *ablactation diarrhea* (*weaning diarrhea*) was much more justified and its origin from alimentary deleterious substances is readily understood because of the inadequate knowledge of the food requirement of nurslings.

The only time when we are warranted in speaking of a physiologic intolerance is upon the first introduction of food into the intestine of the newborn, previously mentioned, and perhaps the colonization therein of bacteria.

The foregoing causes of disease are by no means of like importance. In the development of acute affections we note their frequent combination: those which belong to the second and fourth groups are predisposing, and the

bacterial invasion represents the inciting factor. A certain degree of alimentary dyspepsia is exceedingly common in artificially nourished children; in fact it is almost "physiologically" present. Here also, as Pfaundler has shown, we almost invariably find disturbances in the anatomical function of the stomach which favor the development of abnormal decomposition. As stated above, the bactericidal faculty of the digestive apparatus is lessened, the infection of the intestinal contents or of the walls of the intestine is facilitated. It would seem that in artificial nutrition with cow's milk, which is so liable to undergo change, the numerous opportunities for infection can only be avoided by special prophylactic measures or an unusual resistance to the development of abnormal bacterial decomposition. In some cases no other factors are necessary for infection. It is easily to be conceived that germs, which under normal circumstances exist without injury to the intestinal canal or are destroyed there, may assume the property of accumulation and pathogenic action upon the addition of some predisposing cause.

Furthermore, the artificially nourished infant is much more exposed in consequence of the alimentary damage by chyme infection, so frequently referred to, than the breast-fed child who in this particular is protected by the normal and regular process of digestion. The infectious diseases occur in both categories, apparently with a preference for breast children, although as Moro has recently shown, these possess a stronger bactericidal serum.

The result of these arguments may be summarized as follows: The insight into the microscopic world of the schizomycetes has opened a new era in the previously dark realm of the etiology and pathogenesis of digestive disturbances. We have learned to differentiate two large groups among the great number of bacterial processes running their course in the intestinal canal, one of which by decomposition of the intestinal contents, exerts a *toxic* effect, and the other, by an invasion of the tissue, is *infectious*.

The bacteria of the first group are characterized by an energetic property of fermentation and decomposition; those of the second by pathogenic faculties, also by quite different biologic properties. The typical representatives of the one as well as of the other group permit us to recognize these variations quite distinctly. But even here there are transitions: Original saprophytic organisms or bacteria which are normal in the intestinal canal, such as the bacterium coli or the bacterium lactis aërogenes, take on invasive properties (Baginsky), while typical pathogenic bacteria, such as staphylococci and streptococci, obtain the faculty of acid production to a conspicuous degree. Thus we may observe how both processes follow one another in the same case or appear simultaneously, in that the primary chyme infection leads to lesions of the intestinal mucous membrane and thereby to the entrance of pathogenic bacteria. Here as everywhere we note transitional stages and mixed processes that are sharply distinct from each other in their development and nature. Nevertheless we must hold to this differentiation, which is applicable in most cases, at least at the onset of the disease, as it possesses



not only a theoretic value but is of eminently practical importance for the proper treatment, as the following pages will prove. First, however, a few words must be devoted to the reaction and the changes produced in the intestinal wall by the entrance of these deleterious agents.

The diversity of the irritants which act upon the intestine might lead us to expect as great a variation in the symptoms. As a matter of fact, however, there is a relatively uniform picture. Substances having a toxic effect, such as acids, alkalies, gases, increase the excretion of intestinal fluids and produce an excessive peristalsis, whereby the deleterious products are diluted and removed. Moreover, the epithelium is protected by a tough mucous coating which—as Schmidt has shown—in its natural condition is very resistant to chemical agents. Only in the severest intoxications such as resemble cholera infantum, is there an excessive increase of this mucus, which then leads to extensive degenerative changes of the epithelium. Inflammatory phenomena are absent or at most quite subordinate (Heubner).

In the infectious processes we observe the same symptoms, with the difference that here the inflammatory reaction (hyperemia, loss of epithelium, accumulation of round cells in the interglandular tissue and in the submucosa, etc.) appears earlier and is more distinct. To this must be added the demonstration of the pathogenic agent in the intestinal wall and the elective localization of the changes, which sometimes predominate in the glandular elements, at other times in the stomach or gastro-intestinal mucous membrane.

## SYMPTOMS

The symptomatology, in so far as it relates to the composition of the discharges, has from ancient times been divided into three stages which are governed by the intensity of the irritation:

1. **Dyspepsia**, or, more correctly, the *status dyspepticus*: functional disturbances in the process of digestion without anatomical change in the intestinal wall; characterized clinically by the great residue of undigested or decomposed food.

2. **Catarrh**: a peculiar transformation (obstruction by mucus, or edematous infiltration) of the surface of the mucous membrane, accompanied by mild inflammatory phenomena and conspicuous functional disturbances. Clinically this is characterized by a decided increase of intestinal secretion profusely admixed with mucus. Following the example of West and others I have subdivided this large group, contingent upon its resemblance to the preceding or succeeding stage, into *simple*, *dyspeptic*, *alimentary*, and *inflammatory* catarrh (*Wiener klin. Wochenschrift*, 1901).

3. **Inflammation**: more or less severe anatomical lesions of the intestinal wall, from the loss of epithelium and dense small-cell infiltration up to total necrosis of the superficial layers, fibrinous exudation, ulceration, etc. The feces are characterized by numerous blood- and pus-cells, tissue elements, etc., and the manifestations of inflammatory catarrh.

It is probable that in the not far distant future new methods will arise which will bring the entire symptom-complex into a more sharply circumscribed, etiologically rounded, pathologic picture, as has already occurred in the group of infectious intestinal diseases. At this time, however, there is no other view-point at our disposal for a general division of gastro-intestinal affections.

As a rule chyme infection represents the mild, intestinal infection, the severest type of this gradation. We frequently note transitions of the same disease from one to another stage in a backward or progressive direction, and we are in fact only dealing with a symptomatic division which gains its qualification—alimentary, toxic, infectious—from the etiologic standpoint.

A further and equally important detail in the classification of gastro-intestinal affections is the localization of the disease in the intestine. Although this is often impossible in very young children and when the disturbance is protracted, nevertheless in most cases which are observed from the onset, the part first affected can be recognized; thus, in certain infections of the stomach or the terminal intestine, the upper portion of the bowel is the preferred point of attack with a milk diet, and the lower portion when starchy food has been given. Often quite important data for the localization of the catarrh may be obtained from the clinical symptoms and from the composition of the discharges. In this connection I refer to the excellent exposition of Nothnagel in his *Beiträge zur Physiologie und Pathologie des Darmes* (Berlin, 1884). The objections which have been raised to the views of this author by A. Schmidt do not have reference to nurslings.

This differentiation is not merely of theoretic and clinical interest, but it has a decided influence upon the proper treatment, by medicaments and locally, which, in the gastro-intestinal affections of infancy we must regard as one of the most valuable newer attainments of therapy.

## TREATMENT

Although the discussion of treatment is to be confined to general fundamental laws which are applicable in bacterial gastro-intestinal disease, nevertheless, on account of the intimate relation of all digestive disturbances, the other forms cannot be passed over in silence, especially the dyspeptic conditions which are of primary importance in the development of chyme infection.

As has already been explained in the description of the volumetric method, artificial nutrition itself produces a certain injury to the digestive apparatus, perhaps less on account of the chemical differences in the individual nutritive substances as compared with breast milk, than from the improper administration (influx instead of sucking), and from the over-feeding which is so difficult to avoid. Therefore I attach the greatest importance to the quantitative estimation of the individual meal and of the daily amount, as well as to the limitation of food to the physiologic requirement (minimal diet of Biedert). Observance of these rules will prove the best prophylaxis for diges-

tive disturbances. Even in the more immune breast-fed children profuse and irregular drinking not infrequently leads to disease; how much more so then in artificially nourished infants who are subject almost to a "physiologic dyspepsia."

If these slight disturbances are not arrested by removal of the cause the bactericidal property of the bowel is weakened and infection becomes possible. In children nourished with cow's milk all of the methods must be investigated which are operative in sterilization, in cleansing the bottles, nipples, etc.; even in breast children it is necessary to avoid the adherence of bacteria to the nipple or their permeation to the milk ducts.

Cleanliness of the oral cavity is always to be observed. The influence of the *oidium albicans* in the development of digestive disturbances is still underestimated. To overcome the objections of mechanical cleansing of the mouth I have advised the employment of a boric acid nipple (1899). A number of similar methods have since been proposed. In those cases in which this nipple is rejected I permit the infant to suck a brush dipped in a silver nitrate saccharin solution, as proposed by Concetti.

In an alimentary catarrh, no matter where localized, the following points are to be considered in the treatment:

1. **Removal of Harmful Masses in the Intestine.**—Wherever the procedure is possible (stomach, large intestine) this is accomplished most thoroughly and gently by irrigation; in the small intestine by laxatives, castor oil, or the old reliable calomel 0.01—0.02 per dose, two to four powders taken at intervals of three hours until an effect is produced.

2. **Relief of the Abnormal Decomposition.**—The most obvious measure, destruction of the bacteria by disinfection, is inefficient or impracticable in the intestinal canal. Irrigation of the stomach or intestine may be followed by weak disinfecting solutions or the internal administration of antiseptic remedies, particularly preparations which split up in the intestinal canal. Much less dangerous and more effective is the combat of the processes of fermentation by the withdrawal of those foods which have provoked this condition, therefore, of carbohydrates and particularly sugar in the acid fermentations of the upper intestine, and of albumin foods when the decomposition occurs in the colon with symptoms of putrefaction and alkaline reaction.

This principle is most sharply defined in the employment of Demme-Epstein's albumin water—discovered empirically—for digestive disturbances occurring in milk nutrition. The water appears to alleviate the marked irritability of the gastric mucous membrane in certain of these conditions which are but slightly influenced by the small amount of albumin or by the peptones or albumoses advised by me. As this mixture is rather difficult to prepare, a simple water or tea diet is usually preferred. Marfan states that the child should be given the same amount of other fluid as it would take normally of milk. The complete withdrawal of milk, even in breast-feeding, is the first and most important rule in every alimentary catarrh. This is usually con-



tinued for twenty-four hours in mild cases, but in breast children the period may be shortened. When there has been excessive over-feeding the milk is withheld until the complete disappearance of undigested food from the feces. Strongly diluted food may then be instituted experimentally and cautiously. Whenever possible it is best to begin with a diet which differs from that formerly employed. If the disease appears in milk-fed infants even in the first months I substitute weak Nestlé's or Kufeke's food (one teaspoonful to fifteen teaspoonfuls of water), inversely, in starchy dyspepsia greatly diluted milk is administered. In either case we may attempt to establish a diet corresponding to the age of the patient by the cautious addition of milk.

In the putrefactive processes which run their course in the lower portion of the intestine this principle of deprivation is not so satisfactory, for even with a complete hunger diet the readily decomposable albumin of the intestinal secretion is still present. Therefore in these cases a second method must be invoked which belongs to the so-called principle of bacteriotherapy. Likewise as putrefaction of milk is prevented normally by the presence of sugar, the administration of carbohydrates may combat the existing proteid decomposition in the intestines, since they produce a conspicuously acid reaction and are only absorbed in the lower portions of the bowel. Therefore a diet is required which contains small amounts of starch (Kufeke's infant food) or dextrin,<sup>1</sup> as in Liebig's soup or in the most recent modification of Keller's malt broth. Enemata of starch have an analogous action.

The same method is followed by de Jager, who advises sour buttermilk in the nutrition of children suffering from intestinal disease to prevent putrefaction. In chronic intestinal catarrh, wherein large numbers of proteus bacilli pervaded the evacuations, Brudzinski administered, by my advice, bouillon and whey cultures of the bacterium lactis aërogenes, with the result that the disagreeable fecal odor and the proteus microorganisms completely disappeared from the feces.

**3. Care and Support of Function.**—Conformable to general therapeutic axioms, the diseased organism must be spared as much as possible. This rule has already been observed by the diet of tea and later by the high dilution of the food. Also, in consonance with the fundamental laws of dietetic therapy, we must consider the varying tolerance of the different foods. Albumin and fat are badly borne by the irritable mucous membrane of the intestine. Carbohydrates are better tolerated and the mucilaginous preparations such as salep, rice, and rolled barley, are preferable. The digestive function may be directly aided by the administration of ferments which are lacking (pepsin, hydrochloric acid, pancreatin, Tipe).

**4. Drug Treatment.**—The fourth measure relates to all those drugs which oppose the pathologic condition of the intestine, in that they soothe or stimulate the mucous membrane by relieving pain and influencing peristalsis and secretion. I shall mention only a few examples. One of the most disagreeable symptoms of acute gastro-intestinal disease is the prolonged vomit-

<sup>1</sup> Is changed into sugar too rapidly.—EDITOR.

ing, which exhausts the patient and prohibits the ingestion of fluid. In addition to complete rest of the stomach and the cautious administration of particles of ice most valuable aid has been obtained from creasote, menthol, and cocain.

After evacuation of the intestinal canal I employ the old reliable bismuth, preferably the salicylate (5—100), and when there is colic or permanently increased peristalsis I do not hesitate to give small doses of opium, one to two drops of the tincture to 100 of the mixture, or enemata of a corresponding dose. More caution is necessary in the administration of atropin sulphate, which I have employed since 1890 (Inaugural Dissertation of Weinbuch, Munich) in profuse serous discharges for the inhibition of secretion, occasionally with remarkable results.

R/.

Atropin sulph.....0.01—10

Sig.: One or two drops in fifty of fluid, to be taken in the course of the day.

Dobroklowski has furnished experimental proof for the action of this drug (cited by Lukjanow, *Pathologie der Verdauung*, 1899). When the remedy is administered we must be mindful of dilatation of the pupil,<sup>1</sup> increased pulse, erythema and sudden rise of temperature, which are common consequences; in no case has there been a serious result. Only in subacute and chronic affections do I employ astringents, of which there are various tannin combinations.

A further discussion of indications and the treatment of the individual symptoms does not come within the scope of these general considerations. From the foregoing it will be seen that we possess remedies which will effectually counteract and abort the abnormal processes of decomposition by the rapid and thorough removal of the deleterious contents of the intestine and by preventing the further entrance of fermentative material. If the results are nevertheless incomplete, this is because the physician rarely has opportunity to observe these cases from the onset, but is usually consulted after anatomical lesions and permanent disturbances of function have occurred. This gives time for the cultivation of the ground upon which chronic catarrh with a terminating atrophy, relapses, infectious catarrh, and, finally, severe inflammatory conditions develop. Death is only too often the outcome of these affections, which at the onset are usually harmless and relatively easy to combat, and is an especially common termination when the hygienic conditions are unfavorable.

The circumstances are quite different in the second group of bacterial diseases, the *intestinal infections*. Here the physician is almost helpless in the developed disease. The abortion of the process by laxatives or disinfecting remedies is hopeless in the present state of our knowledge. Although a cer-

<sup>1</sup> Which is not the first or most prominent effect of belladonna at this stage. Flushed cheeks appear first.—EDITOR.

tain influence may be gained by antiseptics over the bacterial processes going on in the intestine, nevertheless we cannot consider a disinfection in the sense of destruction of certain or all of the pathogenic agents. Neither are we able, by passive immunization or the introduction of alexins, to increase the resistance or the bactericidal property of the body, although such an attempt has been made in diseases produced by bacteria of the coli group. The infantile organism must therefore depend upon its own powers.

However, not so much the reaction comes under consideration which is brought about by ejection of the noxious agent or by depriving it of power, as the bactericidal properties and the local or general immunity which follows the infection. We know very little of the factors which are operative here, but next to the inflammatory leukocytic wall, the marked increase of intestinal secretion, irrespective of the intake of food, is certainly a most efficient protective measure. The characteristic bactericidal properties have been only recently discovered. In acute cases independent bacteriologic investigation of the feces reveals a much diminished permeation of the mucous fragments by bacteria, which are grouped in small clumps or zooglae, I might almost say agglutinated. In contrast to this there is a more uniform permeation of the fecal masses, a more profuse and qualitatively different vegetation. In chronic exhaustive diarrheas I have seen conspicuous bacterial decomposition and a fluidification of the mucus, with brownish, soup-like stools, which are probably the evidences of complete destruction of the bactericidal power of the organism. The course and termination of these affections will depend essentially upon the biologic property of the individual pathogenic agent. In one case the toxic, in another the infectious symptoms will preponderate; as a rule, however, the pathologic picture will be of fulminant type from the onset and the entire organism will be involved much more extensively than in the preceding group.

In the absence of specific remedies the treatment must be purely symptomatic. First the intestine must be emptied, provided such a result has not been already accomplished by vomiting and diarrhea. In any event I cautiously employ castor oil, either with or without resorcin, and avoid irrigation of the stomach and intestine. Calomel should not be administered as it produces irritation. The evacuation of the digestive organs and the brief withdrawal of food permit the intestine to concentrate its entire power upon the destruction of the pathogenic agents. Special stimulation of intestinal secretion by laxatives, advised by Kohlbrügge, is uncalled for. A most important object of treatment, however, is the maintenance of a quantity of fluid sufficient to play upon the lumen of the intestine, as is provided by nature. As the ingestion of liquids is prohibited by vomiting, the subcutaneous injection of normal salt solution must be employed.

Here as in all infectious diseases our most important aim is to maintain the natural resistance of the body. Therefore, after a short interval of abstinence from food, corresponding to the condition of the intestine, we must administer stimulating nourishment (meat broth, beef tea, gelatin, wine whey,



etc.). In affections of the colon those foods which contain starch (Liebig's soup, etc.) are preferable because they diminish the danger of decomposition of the secretion.

The severity of the constitutional phenomena and the implication of other organs call for careful and individual management of the symptoms. However, the physician will be able to exert but little influence on the decisive combat between the infection and the organism which so often has an unfavorable termination. On the other hand a wide and fruitful field for his activity will be found in prophylaxis.

### PROPHYLAXIS

Specific general infections due to definite, sharply characterized microorganisms from the intestine appear to be rare in infancy, aside from those diseases which occur in general epidemics. Usually in this category of cases we have to deal with infectious catarrhs, which, judging from the pathogenic agents thus far isolated, are commonly produced by septic or pyogenic bacteria, such as are often found attenuated on the surface and in the surroundings of the human body. While in the latter condition they are usually saprophytes, they may under certain circumstances develop pathogenic properties in the sensitive infantile mucosa. This view is favored by the fact that such affections show a preference for crowded habitations and where human ejecta, filth, and moisture abound, briefly, where all of those conditions prevail which favor the increase of bacteria. The microorganisms gain entrance to the body with the food and saliva by the common use of eating utensils or by the hands, perhaps also with the inspired air, and find their way into the intestine, so that with a certain degree of justice we may speak of miasm. Milk is often the carrier of infective bacteria from diseased cows or from the contaminated atmosphere of the stable, as is obvious from the presence of streptococci in the milk. The staphylococcus enteritis of breast-fed children must also not be overlooked. The most important and dangerous source of these infectious products, however, is furnished by the fecal discharges of the sick. These abound with pathogenic agents which have a conspicuous infective power, and under suitable external conditions give origin to group diseases by contact infection.

The noncommunicability of cholera infantum has long been considered a peculiarity in characteristic contrast to cholera Asiatica. Since, however, the attention of the profession has been directed to the epidemic distribution of cholera infantum the number of such observations has rapidly increased. The extensive investigations in the Foundling Asylum of Prague (Epstein), the reports of Finkelstein from Berlin, of Berton from the Graz Clinic, have all shown that a determining influence upon the morbidity and mortality of foundling asylums is exerted by these affections.

The methods of distribution of this hospital infection depend primarily upon the nurses who care for the children and remove their soiled diapers.

The infectious principle may find its way from the nurse into the food, the nipple, or directly into the oral cavity of the child. But the numerous reports of hospital epidemics show that infection may be transmitted also by means of the child's hands, the diapers, thermometers, instruments, or the commode.

This gives a basis for active prophylactic measures. Airy, dry rooms, avoidance of crowding, careful nursing, and scrupulous cleanliness are necessary for the welfare of the child not merely for general hygienic reasons but also to prevent the development of infectious intestinal catarrh. The nursing provides an exceedingly sensitive reagent for the presence of pathogenic organisms, and in this connection the intestinal mucous membrane does not hold an exceptional position. Therefore, as I have already explained in another place (*Deutsche med. Wochenschrift*, 1898) there must be a certain degree of asepsis in the child's surroundings analogous to that of the obstetrician or surgeon, and experience has already taught that by this simple means we may bring about a betterment of the results.

A further principle that must be observed in our children's hospitals, infant asylums, and in fact in all places where a great many children are assembled, is to prevent the introduction and distribution of infection. Suspicious cases should be retained in an observation ward, sick children should be isolated from the general wards. As we are dealing with a fixed and readily destructible contagium, isolation in a separate space and with a well-trained attendant is sufficient, or a specially instructed nurse alone may be required.

Although we thus succeed in preventing the introduction of such diseases from without, there remains the danger of development in the hospital patient himself, as in institutions an infection by pathogenic germs is sometimes wholly unavoidable. Therefore care must be had that in infectious intestinal disease the germs are not disseminated, that they do not find their way from the ejecta into the food or the oral cavity of the healthy. A most excellent proposition is that of Heubner, who requires a special nurse for the administration of food, and a second to remove the soiled clothing and cleanse the child. Unfortunately, in institutions this suggestion is as impracticable as the requirement that every infant should have a special nurse. In hospital practice we must be satisfied if we have one nurse to every four children, who carefully disinfects her hands after handling a patient, and that every child has its individual thermometer, bottle, nipple, etc. For several years we have had in my clinic small cases made of enamelled ware, with doors of glass, fastened to the wall at the head of the bed, which contain the bottle, a few nipples, clothes, the thermometer, etc., of each patient. This contrivance has proved very serviceable. After an epidemic a thorough disinfection of the ward is necessary, preferably with formaldehyd.

Although these methods fall far short of the theoretic requirement, nevertheless we can observe with some degree of satisfaction that even their accomplishment has produced a noteworthy improvement in these conditions. Above I have called attention to the surprising decrease of mortality since the introduction of these protective measures in the Foundling Asylum in Prague, and

in the two clinics which first chronicled these hospital epidemics. Since the year 1898 there have been isolated group diseases but never again such a great mortality among infants as occurred in the epidemic of that year (*Jahrb. f. Kinderhk.*, LII). We have every reason to suppose that this is the result of the avoidance of contact infection which has been brought about by objective protective measures, and I do not doubt that the introduction of improved methods, such as have been planned for children's hospitals hereafter to be constructed, will produce even better results. Thus we see that these hypothetical conceptions of the pathogenesis of digestive disturbances, even before they have undergone exact scientific proof, have attained practical results which incite us to pursue this method still further.



# CHRONIC DIGESTIVE DISTURBANCES OF INFANCY

By B. BENDIX, BERLIN

IN the course of his career the physician encounters many forms of chronic digestive disturbance in infants. Their comparison, one with another, proves them to be entirely different in their etiology, their symptoms, and their course, or clinical pictures are presented which, although showing many deviations, are readily recognized as types of the same affection. The minority of these disturbances occur in breast-fed children, the greater number being observed in those who are what is commonly termed bottle-fed.

The irregular course, the varying clinical picture, the incalculable outcome of these diseases and the commonly ineffectual therapeutic endeavors, particularly when contrasted with the acute digestive disturbances of this age, makes it obvious how manifold is the relation and how frequently difficult the prognosis of these chronic nutritive affections. Only by repeated investigation and comprehensive study will it be possible to acquire an accurate knowledge of the various forms of this disease, which for many reasons are important and interesting.

It appears advisable, therefore, to review the individual types of these important infantile disturbances and to arrange them into groups.

First, it is necessary to call attention to the *breast-fed* children who for weeks present the symptoms of chronic digestive disease.

Cases are now and then observed of infants who pass thin, greenish-yellow or green stools, immaterial whether taking the milk of the mother or of the wet-nurse. At first, or intermittently, the stools may show the normal yellow color. Frequently they are lumpy and interspersed with small particles. Their reaction is very acid; an admixture of mucus is rare. This deviation from the normal feces is usually a subacute condition and occurs without alarming symptoms. The number of fecal evacuations need not exceed the norm (one, two, or at most three daily); occasionally there may be four or five, but without any increase in quantity, as is the rule in acute gastro-intestinal disturbances, especially in infants nourished with cow's milk. In some cases this dyspeptic stool occurs in immediate connection with, or as a continuation, so to speak, of the dark meconium of the first days following birth. More commonly the movements for some weeks are normal, the disturbance arising later. Vomiting seldom occurs; fever is absent. Examination of the

abdomen reveals neither pain nor meteorism. An intertrigo of the skin readily develops around the anus and upon the upper thigh in consequence of the excessively acid reaction of the feces. The disposition of the child does not appear to be altered. The weight increases normally. Only in rare instances, when the affection is much protracted, is there an actual nutritive disturbance with an irregular or slow increase in weight or even an actual arrest of growth. This

### CHRONIC DYSPEPSIA OF BREAST-FED CHILDREN

does not, as a rule, interfere with the constitutional condition and development. The only symptom that brings the child to the physician is the greenish fluid stool, which continues for weeks.

In the majority of cases the cause of this abnormality is very difficult to discover. The mother or the wet-nurse is perfectly healthy, the interval between the feedings is observed according to the physician's advice, and the quantity of food is not exceeded. An examination of the milk reveals nothing further than that the child may be receiving *colostrum milk*, which we know by experience may cause stools of this character. Under some conditions colostrum may be secreted beyond the normal period, particularly if the breast is not completely emptied. This is usually the case when the infant is too feeble and its power of suction too slight to overcome the resistance of the powerfully developed muscles of the nipple ("schwergehende Brust") and sufficiently withdraw the contents of the breast. Digestive disturbances may then arise. With experience this may be avoided, as the breast that flows freely ("leichtgehende Brust") is of importance in the choice of a wet-nurse for a feeble child. In case this requirement cannot be fulfilled—and in a large city we are often fortunate to obtain any wet-nurse—the "tense" breast of the nursing woman should be given first to a strong child to empty in part, and then to the feeble child.

Only rarely will colostrum be found to have an etiologic significance. In those cases in which it plays no part the source of the chronic dyspepsia will be very difficult to determine. It would be necessary to assume that these dyspeptic infants suffer from a congenital feebleness of digestion, which is borne out by the observation that a child of another family, who is subsequently nourished by a wet-nurse who was discharged because of the dyspeptic condition of the former baby, will thrive and pass a perfectly normal stool.

The *prognosis* in this simple dyspeptic disturbance of breast-fed infants is always good, although in exceptional cases a temporary arrest of weight may occur. The condition may last for weeks or even months—a circumstance that is not so remarkable when we reflect that the etiology of this disease is obscure and we therefore possess no positive indications for treatment.

As the cause of this disturbance, in the majority of instances, is to be sought in the child itself, the attempt to transfer the infant from the mother or wet-nurse to another woman will prove useless, particularly as it is uncer-

tain whether the second wet-nurse will be an improvement over the former one. Nevertheless, the mother will not be comforted with the statement that the dyspepsia of breast-fed children is not serious, and therefore a rational treatment of this slight disturbance must be attempted. For this purpose a careful investigation of the nurse's general condition, physical as well as mental, and of her digestion is necessary, besides a control of the size of the meals and the intervals between them. Any irregularities on the part of the nurse or mother must be corrected; improper distribution of the food must be regulated. If the feedings are too frequent the interval should be lengthened, perhaps to four hours between the meals.

In some cases examination of the milk reveals an excessive percentage of fat. To overcome this condition the breast-milk may be diluted by giving to the child, before it is placed at the breast, 50-75 grams, according to age, of fat-free bouillon or gruel decoction.

If it is thought that most of the cases have to do with a congenital decrease of the secretory function of the infantile digestive system an attempt should be made to strengthen the digestive activity by the administration of stimulative or even fermentative remedies. As a matter of fact a speedy improvement often follows the so-called *ferment therapy*. In the order of their digestive action the following remedies must be mentioned: pepsin (as much as will go on the tip of a knife), lactopeptin, 0.15-0.25 grams, and pankreon, 0.25-0.3 grams. According to the severity of the case one or another of these drugs is given after each meal or every second meal in a small quantity of breast-milk. With pankreon particularly I have often seen a complete cure in a few days. Good results are sometimes obtained by purgatives, best by 01. ricini ( $\frac{1}{2}$  to 1 teaspoonful) given for a few days in succession or occasionally at intervals, or, after cleansing the intestine, the ferment treatment is employed, perhaps in the form of a simple muriatic acid mixture.

Only in extreme cases, where these therapeutic measures prove ineffectual and regulation of diet, the relief of constipation or other irregularity on the part of the mother or wet-nurse are without avail, it is well to wean the child or change the nurse. This should be delayed as long as possible, as even under new conditions there is no guarantee of success.

In contrast to those infants whose only symptom consists of an altered appearance of the feces, there are breast-fed children who present the clinical picture of *atrophy*, common with artificial nourishment, which will be described more fully later.

Here we are dealing with infants who are pallid and emaciated. The skin is pale or of a grayish tint, the weight has not increased for weeks or may even have diminished. The bowel movements of these infants are either sluggish, constipated or dyspeptic, or the last two conditions may alternate. These nurslings, all of whom present the same miserable habitus of the body and the same care-worn expression, have usually been nursed by the mother; only a small minority have been brought up by a wet-nurse. The etiology



and the history elicited from the relatives of the little sufferer usually coincide word for word.

The mother nurses her own child for two or three weeks; everything goes well, then there is an arrest of weight. This arrest, occasionally with an insignificant increase, continues for weeks. At first but little importance is attached to it, or with a slight increase in weight the anxiety is removed, but eventually the profound gradual change becomes obvious to the mother, especially if the disturbance be associated with some anomaly in the function of the bowels. If the mother of such a baby is questioned as to the condition of her milk she will usually reply that the supply is sufficient or that it is so abundant that a quantity is discharged in the intervals between the feedings; or, as has occurred with me several times, the physician is consulted about a breast-pump with which the mother may reduce the excess of milk.

In the failure to recognize these conditions lies the serious error of the mother. Without the slightest suspicion of the fact, her child suffers from hunger and gradually passes into a condition of invalidism. The proof of this may be readily demonstrated by comparing the daily quantity of milk secreted by the breast with the quantity necessary for a normal child of the same age.

The method by which the quantity of milk secreted by the breast is determined is of great practical value in the various disturbances arising in the nutritive therapy of infants. It can be utilized upon many occasions and in addition to many clinical results it is the only positive guarantee as to whether the breast supply is enough, too much, or too little. An opinion regarding the amount of milk withdrawn can be obtained by *weighing the infant* before it is put to the breast, and again when it has completely satisfied itself. The difference in weight, notwithstanding the loss by perspiration, gives a sufficiently satisfactory idea of the quantity of milk, that is, of the capacity of the breast.

## ATROPHY OF BREAST-FED CHILDREN

If the weighings are taken after each meal for the twenty-four hours a decided deficiency will be found in the quantity of the milk compared with the normal amount necessary for the growth of the infant.

In some instances the secretion of breast-milk is so slight in comparison with the normal quantity that weaning at once becomes necessary; in other cases it is but little less than the norm.

Figure 1 shows the very characteristic curve of atrophy in a breast-fed child (C. P.).

This child, which was nursed by a mother with an apparently profuse flow of milk, within four weeks from birth gradually declined from the original weight of 2,575 grams to 2,510 grams, instead of increasing during this period about 600 grams. Upon the first of August (the twenty-ninth day of life) the quantity of milk determined by weighing amounted to 335 grams, or about one-half of the normal. The addition of a 300 grams bottle of one-

half milk at once supplied the deficiency and was followed by the ordinary increase in weight. It is of interest to note that the mother, whose daily supply of milk was about 400 grams was able to nurse her child for five months longer.

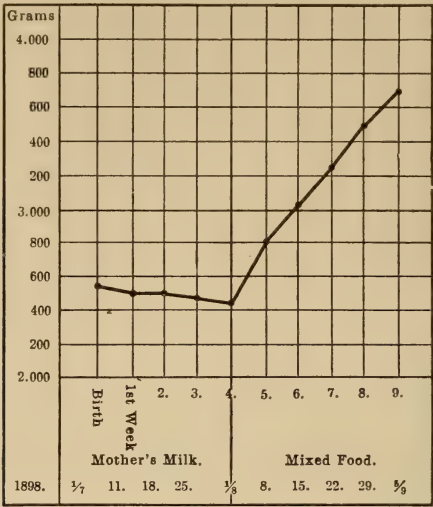


FIG. 1.—Breast-fed child, C. P. Atrophy.

		Weight		Difference.
Date.	Hour.	before nursing.	after nursing.	
1899.....	1/8	4	3490 3535	45 grms.
"	"	7	3420 3460	40 "
"	"	10	3450 3480	30 "
2/8	2	3430	3480	50 "
"	7	3440	3530	90 "
"	10	3500	3530	30 "
"	1	3450	3500	50 "
		In 24 hours.....		335 grms.
"	4	3450	3500	50 "
"	7	3490	3520	30 "
"	10	3120	3220	100 "
"	1	3230	3250	20 "
"	1	3440	3490	50 "

With the demonstration of an insufficient secretion of milk, i. e., of a total lack of milk in the breast, the etiologic factor of infantile atrophy is found, provided we are not dealing with the previously described disproportion between a "tense" breast and a delicate child.

The diagnosis at the same time indicates the method of treatment. The only requisite is to supply a quantity of food proportionate to the age of the child (perhaps also to the weight) by the addition of some artificial food. How much is to be added is readily determined by comparing the deficiency

in weight after the feedings with the normal amounts for healthy breast-fed children.

These normal values, which have been obtained from the valuable researches of many months by Ahlfeld, Bendix, Feer, Hähnel, Pfeiffer, and others, give us an exceedingly clear idea of the requisite and needful amount of fluid for infants, and therewith under abnormal conditions a measure for the plus or minus quantity in the individual case and a guide for artificial nutrition.

Nevertheless these figures must not be regarded as an unalterable scheme; on the contrary, it must be remembered that every individual is subject to the function of his digestive apparatus, to the activity of the glands and their varying secretions; consequently one person will satisfy his economy with a smaller amount of food than another, yet both will present the same percentage of gain. Thus, it is not remarkable for one baby to require more food, another less than is indicated in our calculations. In all cases, however, a brilliant and immediate cure will result if the insufficient natural food is reinforced by the addition of cow's milk.

In marked contrast to these atrophic breast-fed infants there is another group of dyspeptic symptoms which have been acquired from too frequent or too profuse feeding; in other words from a surfeit of food. In a very few weeks these infants present the typical picture of

## OVER-FEEDING OR HYPERNUTRITION

The mother believes the child to be remarkably well-developed and unusually healthy, since the weight far exceeds the norm; it is the so-called "model baby," the "mother's pride," surpassing in weight all other infants among the relatives and friends. But this happiness of the mother is short-lived. Superfluous fat soon appears, the entire body and especially the face has a swollen appearance, the complexion is pale and yellowish, the abdomen is distended and tense, and colic is common. In addition to these symptoms there is often a certain restlessness, which appears early and may cause an increased excitability of the motor and sensory nerves (facial phenomenon, timidity, etc.), occasionally reaching its acme in eclamptic or tetanic attacks.

The condition of the bowels in these infants, at first normal, is sooner or later changed. Either there is the typical picture of *enteritis*, the feces being voided in small quantities admixed with mucus and accompanied by tenesmus, or with hypernutrition the feces reveal the picture of *fat diarrhea*; the discharges are bright yellow, homogeneous or permeated with whitish particles, somewhat thinner than normal, and with a distinct gloss of fat, frothy, caudle-like. Microscopically fat globules with the glistening appearance of mother-of-pearl, preponderate in the feces in large drops and in clumps. In addition scant or profuse quantities of fatty acid crystals are sometimes noted, mostly arranged in layers and pointed like blades of grass, but also in the more plump, stellate, crossed arrangement or even in amorphous yellowish clumps of fatty calcium salts.



The diagnosis, which to the experienced physician is apparent from the general condition and from the feces, is also revealed to the tyro by the result obtained by the scales regarding the amounts of milk consumed. In how far the normal quantities of milk are exceeded is shown by Figure 2.

**Treatment.**—The treatment of these cases is simple and mostly brings

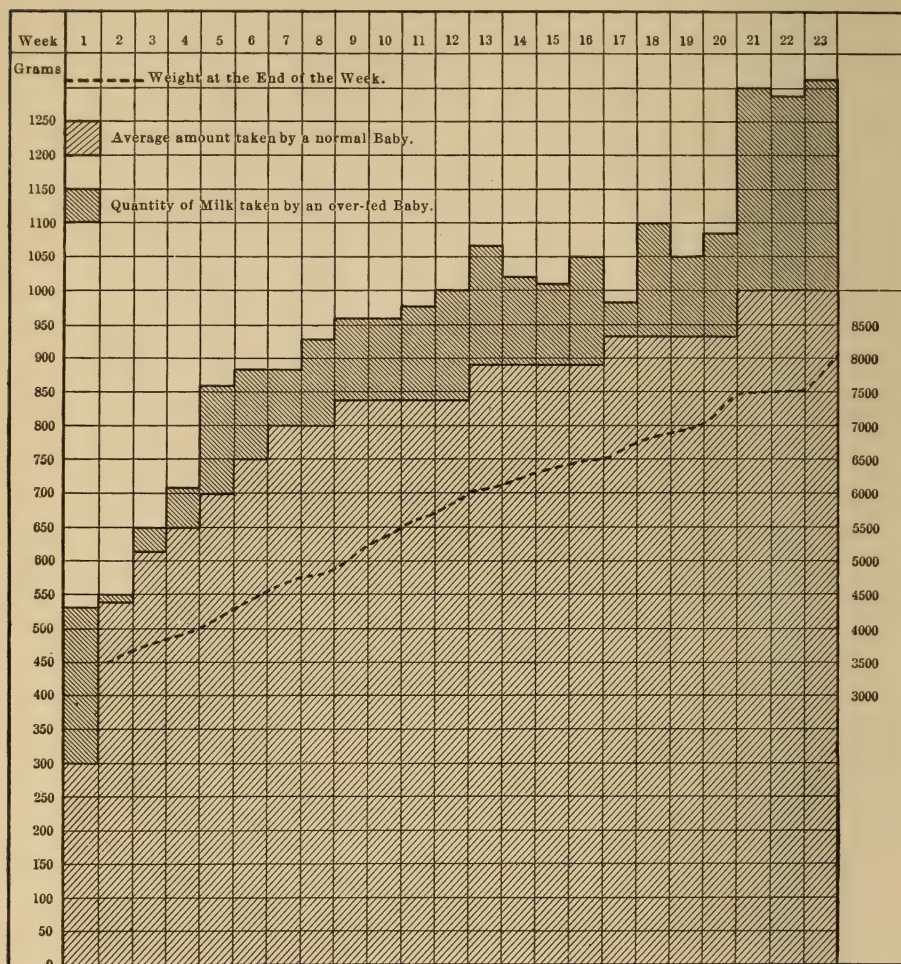


FIG. 2.—Infant E. K. Hypernutrition.

about a relief of the symptoms and a cure. The bowel is first cleansed by the administration of a purgative (calomel 0.03, four doses; pulv. Magnes. c. Rheo, pulv. glycerh. comp. 1-2 grams, or Ol. Ricini 3-5 grams. The success of treatment, however, depends upon a careful regulation of the feedings, which must not be too short, nor should the child be allowed to completely satisfy its hunger, thus preventing an excess of nourishment. There are two

ways of meeting the last requirement: the child is either kept at the breast a shorter time, say ten or fifteen minutes instead of twenty minutes; or the weight is taken before nursing and again after a fixed time at the breast. The result obtained from the scales is then compared with the figures of normal individual feedings for the same age, and whether the child shall be permitted to nurse for a longer time depends upon whether the figure is too great or too little. After a time a quite accurate estimate can be made without weighing as to how long the infant should nurse without consuming too great a quantity of food.

A second method for the relief of the condition depends upon the administration of a thin mucilaginous preparation or of bouillon free from fat before the child is put to the breast, thus partially allaying its hunger so that it will take a smaller amount during nursing.

Now and then the fatty diarrhea due to hypernutrition does not yield at once to this dietetic regime; then, as in the case of chronic dyspepsia, pancreon is often of great service.

Attention must now be turned to a pathologic condition in which the fulminant and characteristic phenomena present a well-marked symptom-complex, which nevertheless is in such great contrast to the etiology and genesis that in spite of the investigations of many physicians (Finkelstein, Thomson, Knöpfelmacher, Freund) it is yet unsolved and obscure.

### CONSTRICTION OF THE PYLORUS, PYLORIC STENOSIS, SPASM OF THE PYLORUS

In constriction of the pylorus we are dealing either with a genuine hypertrophy of the muscular layers of the pylorus (organic stenosis) dependent upon an anatomical basis, or only with a periodic and transitory spasm of the pylorus with a decrease in its lumen. In the wider sense we include with this congenital anomaly of the pylorus a well-defined stenosis or spasm in other areas of the intestinal canal, especially in the duodenum, but also in more remote parts of the digestive tract (enterospasm). Constriction of the pylorus may be regarded as the type of these pathologic forms, being characterized more than any other similar disturbance by the frequency of its appearance, and impressing its stamp upon the clinical picture by the uniformity and clearness of its symptoms. Whether in the individual case we are concerned with an organic or functional disturbance can rarely be determined with certainty, as the clinical symptoms of both conditions, notwithstanding their unlike basis, differ but little except that the severity and duration of the pathologic process may awaken a suspicion of an anatomical stenosis.

Apart from those cases of genuine stenosis or complete atresia, the outcome of which is speedy death, the practitioner is chiefly concerned with the constrictions of slight grade or the simple recurrent spasms of the pylorus.



Included in this group are a number of cases, mostly breast-fed children, which present a peculiar, unmistakable clinical picture that can be produced only by pyloric occlusion.

**Symptoms.**—The most marked symptom, and one which dominates the condition, is vomiting in a form which is remarkable for its tenacity and intensity. A further characteristic is its appearance immediately after the ingestion of food, or rarely later than thirty minutes or an hour afterward. The milk is usually uncoagulated and is vomited in large quantities, sometimes more than the child has consumed at the meal. According to the degree of constriction and the stage of the disease the infant vomits after every meal or only after a few, or perhaps merely in attacks, the food being retained for a half or even an entire day. The vomitus never contains bile. No matter whether such infants are given the quantity of milk appropriate to their age or whether this is reduced to a minimum, whether long or short intervals elapse between the feedings with a reduction in the quantity of milk, no matter what attempts are made to regulate the diet, all are alike useless in arresting the vomiting; the attacks return anew. Gastric lavage, even when repeated, does not relieve this tumultuous condition of the stomach.

In some instances vomiting appears immediately after birth, in other cases not until days or weeks later, and exceptionally at the end of the second month of life. This well-characterized vomiting, occurring as a symptom of spasm of the pylorus, is always limited to early infancy.

If the vomiting does not appear within the first few days after birth either there are no disquieting prodromes on the part of the stomach or there is merely a tendency to vomit after overloading of the organ with too much food. This latter variety, which etiologically is to be referred to injurious food and dietary errors, is rare and encountered only in bottle-fed infants.

The second conspicuous symptom is an abnormal condition of the bowels. The stool is scant; constipation for days is the rule. In the well-developed disease feces are lumpy, dry, of a dark brown or black color, occasionally consisting of small, firm balls or particles, "chainlike, resembling sheep-dung or coffee beans." The normally soft, pappy consistence which is peculiar to the feces of infants in the first half year of life is entirely lacking. Evacuation of the bowels never occurs spontaneously but results only after the administration of repeated doses of purgatives or after enemata. Even these remedies are often ineffectual.

In the milder cases periods of amelioration alternate with periods of aggravation, but here also the feces are in broken particles or formed and are only passed with the aid of purgation.

On account of the slight absorption of fluid diuresis begins to abate: the infant is rarely wet.

As a rule the appetite is good; very frequently there is eagerness for food and for greater quantities than would be given normally. While healthy infants generally fall into a quiet sleep after nursing, the child with constricted pylorus writhes and cries as if in pain and does not become quiet until



the stomach is emptied by vomiting. Frequent restlessness and occasional whimpering or crying occur also during the intervals between the feedings, therefore at a time when the stomach is empty or contains an insignificant quantity of food. This symptom favors the fact that the cause of these painful sensations, the pathologic contractions of the pylorus, are either permanent or occur at intervals even when the stomach is empty.

Examination of the abdomen of an infant suffering from disease of the pylorus shows the region of the stomach to be prominent, especially after the ingestion of food, and in marked contrast to the retracted abdomen. Occa-

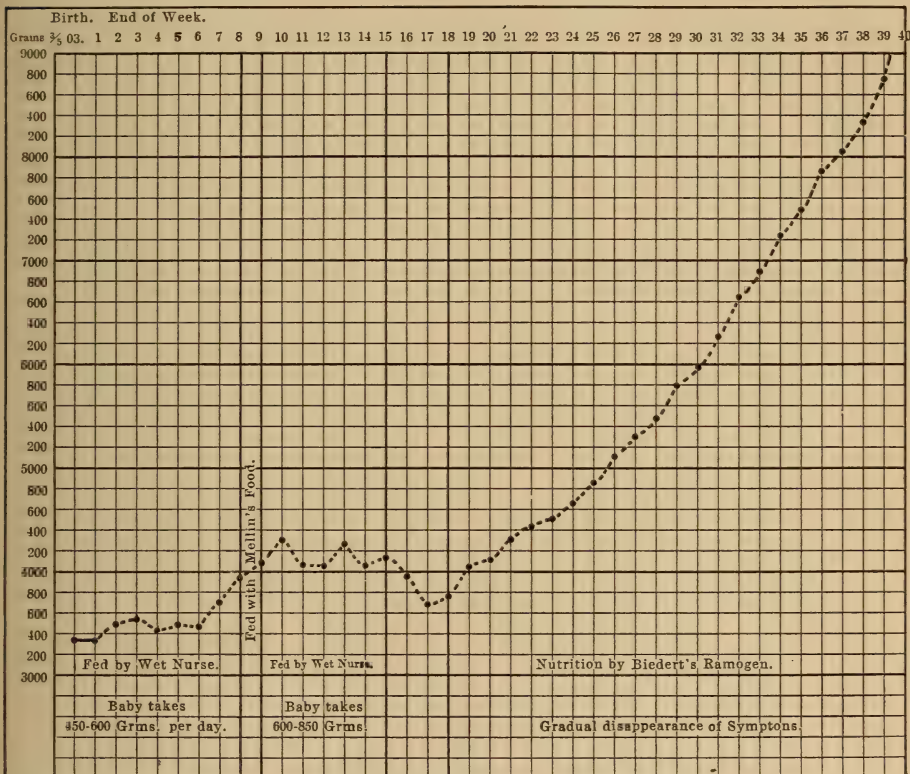


FIG. 3.—Symptoms: stubborn constipation; vomiting of nearly every meal, often immediately after the ingestion of food; antiperistaltic movements; retracted abdomen. Nervous infant. Atrophy. (Rudolph K., born May 3, 1903: spasm of pylorus.)

sionally by sound palpation or by insufflation of air a visible dilatation may be noted and a demonstrable plastic peristaltic movement of the stomach or of parts of the intestine may be determined. These conspicuous peristaltic movements, which often occur over the abdomen spontaneously or may be produced by stroking the belly with the finger or the handle of a percussion hammer, are usually wave-like and run transversely over the abdomen from

left to right; sometimes, however, they are "antiperistaltic," proceeding from right to left.

In some well-developed cases of pyloric constriction it is possible to palpate on the right of the vertebral column a cylindrical, transverse tumor of the thickness of the little finger—the contracted pylorus—or at least a resistance in the pyloric area.

At the onset of this digestive disturbance the development of the child, usually of normal weight at birth, continues fairly good; later, when vomiting dominates the scene and less food is assimilated in consequence of this symptom, there is an arrest and finally a loss of weight which may progress to the most marked grades of emaciation or even to complete atrophy.

The duration of the disease and its final outcome—atrophy—are naturally in intimate association with the degree of constriction and dependent upon whether the condition is one of organic or functional change.

I have seen a bottle-fed baby, aged five weeks, which presented all of the characteristic phenomena of pyloric stenosis but nevertheless was cured of the malady within three weeks. Another case, that of a breast-fed infant, in whom the predominant symptom, vomiting, appeared immediately after birth, was a cause of great anxiety to the parents for many weeks, and not until the termination of the eighteenth week of life was there a permanent gain in weight. (See Fig. 3.)

**Prognosis.**—In congenital anatomical alteration of the pylorus the prognosis depends entirely upon the extent of the stenosis and upon the functional capacity of the expulsive musculature of the stomach. With moderate stenosis and powerful muscles the prognosis is fairly good. The child who finally overcomes the acute perils of a relatively constricted pylorus, which may continue until the end of the fourth year, may in later childhood develop a permanent gastrectasis with all of its annoyances and dangers. In absolute pyloric stenosis the prolongation of life in the individual case is very questionable. Most favorable are the cases of relative stenosis in which there are apparently only recurrent spastic constrictions of the pylorus. These simple, functional disturbances appear in the majority of cases to be the etiologic factor for the severe clinical symptoms, therefore they also furnish the greatest contingent in practice. It is a comforting fact for both the physician and the parents that patients belonging to this category almost always recover, even after the severest atrophy. When the stage of improvement arises, although by what means it is brought about is unknown, it is nevertheless certain that the change is usually quite sudden and as a rule permanent.

The pathogenesis requires amplification at many points.

For many of the cases the question propounded at the beginning of this article as to whether pyloric constriction develops from an organic or functional basis must remain open. In the numerous cases that pass from gradual improvement into complete recovery a transitory spasmodic condition of the pylorus must be assumed; in other instances, in which from birth the severest symptoms occur acutely, with rapid atrophy, a true congenital pyloric stenosis

can scarcely be rejected. As a matter of fact, in a number of children who have succumbed to the disease the autopsy findings render this assumption positive, since they disclose an anatomical foundation as the cause. The necropsy findings and the reports of various authors (Hirschsprung, Finkelstein, Thomson, Stern, Löbker and others) are as a rule conspicuously uniform: The pylorus is usually found as a coarse, hard, cylindrical tumor about  $2\frac{1}{2}$  cm. in length which is separated externally from the pars pylorica by a shallow furrow, and internally by step-like elevations. The lumen of the organ has been variously described, sometimes as admitting only a fine probe, at other times one of medium size or occasionally of the size of a lead-pencil. The mucous membrane at the entrance of the pylorus is deep red and greatly congested; particularly at the point corresponding to the insertion of the small curvature of the stomach the swelling stands out valve-like and prominent, and, like a valve, occludes the passage to the lumen of the pylorus. The stomach is enlarged, the wall, and especially the muscularis, greatly thickened, the mucous membrane is infiltrated, there is polypoid proliferation, and between the glands there is a small-cell infiltration. The hypertrophy implicates all of the tissue layers and in particular the muscularis, sometimes with a greater involvement of its longitudinal, at other times of its annular musculature. Hirschsprung succinctly defines the post-mortem findings of pyloric stenosis as "congenital pyloric hypertrophy" and Finkelstein also considers "congenital pyloric stenosis" to be responsible for a number of the reported cases. The latter author believes the muscular hypertrophy and the gastric dilatation to be secondary manifestations. He analyzes the condition in the following order: congenital pyloric stenosis and in consequence stagnation of the gastric contents; then intercurrent dyspeptic disturbances and catarrhal affection of the stomach; the swelling of the mucous membrane increases the stenosis, the inflammatory infiltration of the walls of the organ paralyzes the motor function and leads to gastric distension. With this there is compensatory and inflammatory hypertrophy of the muscularis.

In contrast to the explanation of pyloric stenosis as a congenital, organic, muscular disease, other authors (Thomson, Pfaundler) have claimed that in the pathologic process a firm anatomical obstruction to the passage does not exist but merely a "spastic" constriction or a "spastic" closure of the pylorus ("congenital" or "idiopathic vomiting" or "congenital gastric spasm," Thomson). Pfaundler regards the thickened, rigid wall of the pylorus, the lumen of which is so narrowed as to scarcely permit the passage of a thin sound, merely as a post-mortem finding, as he was able to demonstrate that in infants who had never presented gastric symptoms during life the entire stomach or its pyloric portion were occasionally in firm contraction.

For the origin of spastic pyloric stenosis various theories have been propounded. Thomson speaks of a nervous disturbance of the coördination activity of the gastric and pyloric musculature so that even *in utero*, during the absorption of liquor amnii, a continuous uncoördinated labor is performed by the stomach; other authors refer this uncoördinated pathologic activity of



the stomach to the influence of a harmful irritation which the infantile organ has suffered *post partum*.

From the investigation of my own clinical material, which has amounted to eleven cases in the last few years, it is evident that in the majority a nervous predisposition has existed from the onset. The infants are restless, frightened, and present the facial phenomenon, not as a secondary condition but primarily, at the beginning of the disease. A number of my patients were descended from nervous families, and a special predisposition existed in the child whose weight curve I have presented; here both the father and mother were exceedingly nervous individuals. If we adhere to the theory of a nervous predisposition it is necessary to assume that through a certain hypersensitiveness and perversity of the nervous apparatus of the stomach the filling of that organ results in closure of the pylorus. According to Köppen, who declares the pyloric spasm to be a congenital, local, constitutional anomaly, the hereditary nervous predisposition plays an important rôle. Also, in conformity herewith is the peculiar phenomenon of a family predisposition to the affection, as has been reported by Henschel, Freund, and others.

A further indication in the analysis of the nature of pyloric spasm is furnished by the verification of Knöpfelmacher of the presence of hyperchlorhydria in an infant suffering from pyloric stenosis. This abnormality of the gastric juice secretion, which occurs in constriction of the pylorus and similar conditions in adults, has also been demonstrated by other authors (Freund) in infants with pyloric stenosis. If this gastric hypersecretion of HCl is found to be a common condition in infants with constriction of the pylorus it would explain why the disease is so common in breast-fed children, for in consequence of the slight amount of albumin in breast-milk there is also a lessened property of combination of HCl, hence a plus of free HCl is present which furnishes the irritation for the spasmodic contraction of the pylorus upon filling of the stomach. This, of course, would only explain the condition in breast-fed infants, and not in all of these, for only in the minority of such children has an increase of HCl been demonstrated. For infants nourished with cow's milk, in which with the large amount of albumin present there is a greater liability of combined HCl this explanation is not sufficient.

**Treatment.**—In the treatment of pyloric constriction I shall exclude those cases in which, from the evidence of the clinical symptoms, we are dealing with a congenital organic or absolute stenosis. For these cases a gastro-enterostomy has been proposed. It is well, however, not to be too eager to advise operation as even the cases of pyloric constriction, apparently the most severe, yield to internal treatment, and an operation is no guarantee of a successful termination. Surgical intervention should be considered in those cases in which vomiting appears immediately after birth and fulminant symptoms with rapid decline indicate a serious prognosis—cases which we are certain will perish if a speedy operation is not performed.

In the majority of patients, however, it is advisable to desist from surgery and to adopt an expectant or internal treatment.

The principal object in a malady in which vomiting is the most prominent symptom must be to find a food that will agree with the infant. As breast-milk, for reasons which need not be mentioned here, is the best and most suitable food, the first thought would naturally be to continue this nourishment and also to supply breast-milk to bottle-fed babies. Ultimate recovery from pyloric stenosis after the administration of breast-milk has been reported by Heubner, Stamm, and others. In a great majority of the cases (in my material 9 times out of 11) breast-milk fails as a remedy for this affection. A portion of my patients were nursed by the mother, others by a wet-nurse. After several changes of the wet-nurse I was reluctantly compelled to substitute artificial nourishment for the natural food because of the persistent vomiting, the stubborn constipation, and the continuing loss of weight.

On account of the hyperchlorhydria now and then present in pyloric stenosis, and of the greatest faculty of HCl combination with undiluted cow's milk, this would be the rational food after failure with breast-milk. Thus, Knöpfelmacher, Freund and Siegert report cures in pyloric stenosis following the administration of undiluted cow's milk. Personally I have not employed this method but with the indication given by the hyperchlorhydria for therapeutic measures, and having failed with breast-milk, I should first consider this experiment, and would recommend it all the more urgently if an increased accumulation of hydrochloric acid were the result. Should this attempt prove unsuccessful I earnestly advise a form of nourishment from which I have seen positive benefit after failure with undiluted and diluted cow's milk, Liebig's soup, and other milk substitutes. This consists of Biedert's cream mixture, which, under the name of ramogen, is supplied by the manufacturer as a conserve as well as in a fresh form ready for use. At first I administer this cream mixture cautiously, in small amounts, increasing the strength and quantity gradually. I have read with much interest that the plentiful employment of cream in the diet has also proved useful in gastric ulcer of adults by counteracting the effect of hyperacidity, and that much may be accomplished by the substitution, so far as possible, of cream for the readily fermenting carbohydrates, particularly in those cases in which there is a tendency to hypersecretion and hyperacidity (Strauss).<sup>1</sup>

With the administration of Biedert's cream mixture more than with any other food it will be noted that the vomiting is gradually decreased, becomes less frequent, appearing only occasionally, and finally ceases.

As to the quantity that is to be given at each meal, in some cases it appears to be practically immaterial, so far as the frequency of vomiting is concerned, whether the child receives much or little. In other cases the patient apparently thrives better with small amounts (20-50 grams per meal) as now and then, with this limitation, a meal will be retained by the stomach.

<sup>1</sup> I do not consider it my domain to criticize, but fat creates fat acid and is not borne in gastric ulcer. Its excess—aye, its quantity appropriate in normal conditions—gives rise to acid, indicanuria, diacetic acid, and acetone. Carbohydrates are not "readily fermenting," just the contrary.—EDITOR.

In these cases, naturally, the intervals of three or four hours between the feedings must be shortened to thirty minutes or an hour. Experience has taught that it is advantageous to re-supply the stomach with food after the vomiting, as then the fluid will often be retained.

Gastric lavage, which is so valuable in the acute gastro-intestinal affections of infancy, not only is of no avail in pyloric stenosis, but through the development or aggravation of gastric dilatation is even a source of danger. Only at the beginning of the treatment may lavage be indicated to free the stomach of milk and fermentative residues.

The following drugs are of service in pyloric stenosis, especially for the vomiting and the spasmodic contractions associated with pain:

R/.

Anesthesin ..... (0.15-0.25)

One powder thirty minutes before each feeding; opium in combination with valerian:

R/.

Tinct. Opii.....gtt. i-ii

Tinct. Valerian.....10.0

M. et Sig. Five to ten drops three times daily; or alkalies.

R/.

Kalii carb.....4.0-6.0

Syr. Cort. Aurant..... 50.0

Tinct. Opii.....gtt. v-vi

Aq. dest.....ad. 100.0

M. et Sig. One teaspoonful after each meal; or

R/.

Magn. carb..... 4.0

Tinct. Opii.....gtt. v-vi

Syr. Alth..... 30.0

Aq. dest.....ad. 100.0

M. et Sig. A small teaspoonful after each feeding; the bottle not to be shaken.

Moist-warm applications or flaxseed poultices applied to the region of the stomach and renewed every two or three hours are valuable for the relief of pain and to lessen the spasm. Frequent warm baths are of value when the pain is intense.

For the constipation, which, next to the vomiting, is the most prominent symptom, and, as has been stated, is very obstinate, enemata of water, oil, or glycerin may be employed, or suppositories, high injections, mechanical loosening or evacuation of the bowel by means of moderately thick rectal



bougies. Abdominal massage may also be cautiously tried. In desperate cases my greatest success has been from the use of rectal bougies. Although purgatives must not be employed regularly, they cannot be dispensed with entirely, even if they are only administered at intervals (about every eight days) to relieve the bowel of the inspissated residues. Most suitable for this purpose is the addition of a few teaspoonfuls of malt extract, given several times a day with the food, or 5 to 10 grams of castor oil.

The constipation regulates itself very gradually; it disappears much later than the vomiting, continuing as a rule for weeks or months and even after an increase in weight is noticeable. Usually the condition does not disappear until the infant is transferred from an exclusive milk diet to mixed food.

Pyloric stenosis has here been described as a pathologic picture of infancy, which, although more common in breast-fed children is occasionally observed in bottle-fed babies. Another chronic digestive disturbance

### CHRONIC CONSTIPATION

is also encountered with both forms of feeding, but is most common in artificially-nourished infants. By habitual constipation we understand a chronic digestive disturbance in which, instead of the normal fecal movements of salve-like consistence occurring once, twice or even three times in the twenty-four hours, the physiologic action is disturbed, in that the intestinal contents are evacuated spontaneously only every two or three days or perhaps not at all. In the overwhelming majority of such cases the consistence of the evacuated masses is compact, lumpy or formed, the color brownish-black, sometimes actually carbonized. While in many of these cases the only symptom is the sluggishness of the intestine or coprostasis, in others the abdomen is tense and meteorically distended. Also the tongue is coated, the appetite poor, and there is a certain amount of restlessness.

Although the diagnosis of habitual constipation is not difficult the *treatment* demands an exact knowledge of the underlying source of the evil. With the etiology the direction for therapeutic procedure is indicated. The causes of chronic constipation in infancy are various and treatment must accordingly be directed to the underlying condition. We have learned that constipation is a symptom of hypernutrition of infants and have likewise noted the condition when insufficient food is administered. It is found under either circumstance in breast-fed as well as in artificially-fed children, although breast-fed infants furnish the great contingent of the latter group and bottle-fed infants of the former. In bottle-fed children the quality of the milk as well as the quantity is often an important factor in the development of constipation: Undiluted milk may occasionally favor this condition in early infancy or, inversely, a too great dilution or too great addition of infant food. Now and then constipation occurs during the slow healing of a chronic intestinal catarrh.

Constipation not infrequently occurs during weaning, that is, at the period when the child is transferred from milk to a semi-solid or mixed diet.

In many instances none of these factors is available and the explanation must depend upon the so-called atony or debility of the intestinal muscles, whether this be congenital or acquired through hypernutrition. As a predisposing factor for this insufficiency the slighter development of the intestinal musculature of the infant may be invoked, and even more important is the anatomical condition of the colon, which, in proportion to the small intestine is much longer in early infancy than in the older child and adult, and at the same time by its greater mobility at the mesentery often discloses a more or less decided loop formation at the sigmoid flexure. We can therefore appreciate why, in infancy, there is frequent difficulty in the propulsion of the intestinal contents, which results in chronic constipation.

In very exceptional cases there exists from birth a stubborn constipation and distension of the abdomen which may from time to time be increased. Upon examination of such patients it is found that thick bougies per rectum may be introduced easily and to some distance. Simultaneously there is a greater capacity of the large intestine and through the abdominal walls a visible dilatation of the colon and a palpable thickening of its walls may be demonstrated. For a time the child thrives very well with this condition; later, emaciation takes place rapidly, usually complicated with profuse diarrhea, and death ensues from inanition or cardiac asthenia. In some instances the patient has been saved by a systematic regulation of the defecations. This affection, which is known as

#### HIRSCHSPRUNG'S DISEASE

because of its first recognition by that author, and of which Genersich, Johannessen, Göppert and Baginsky have since reported cases, in all probability is due to a congenital hypertrophy and dilatation of the large intestine which may be regarded as an anomaly of development, as the conspicuous meteorism and constipation are present at birth. In the cases which have come to autopsy an anatomical thickening of all the layers of the intestinal wall has been found. Besides this congenital hyperplasia of the muscular layers, however, there must be a certain lack of motility and of complete power of contraction to explain why, notwithstanding the hypertrophy, the large intestine is incapable of expelling its contents. Stasis of feces and gas still further increase the hypertrophy and sometimes gives rise to secondary alterations of the mucous membrane (erosions and ulcerations). In the cases which have thus far been reported no constriction could be demonstrated in the lower portion of the intestine at the internal sphincter.

In contrast to the general acceptance of this malady as a *dilatatio et hypertrophia coli congenita* Johannessen declares the condition to be acquired, upon the basis of the previously described physiologic peculiarities of the sigmoid flexure which may under some circumstances in the infant give rise to

difficult defecation and to a labor hypertrophy of the muscular layers, and finally to dilatation of the intestine.

In another series of cases the presence of superficial or deep lesions of the intestinal mucous membrane,

### EROSIONS AND FISSURES

may reflexly produce constipation in consequence of pain during defecation. Although such infants present a very characteristic picture, the condition is often unrecognized by the physician because he omits to examine the anal mucous membrane for injuries. Infants who have always had normal, painless evacuations show that they are in pain by crying prior to every bowel movement, which still takes place spontaneously. This pain continues during or after defecation. Within a few days the sensitiveness during a bowel movement becomes extreme and in order to avoid the pain the child retains the feces as long as possible. The previous restlessness and refusal to attempt defecation are even more significant in children about one year old than in nurslings.

In consequence, if the anal fissure persists for some time, small hemorrhoidal nodules often develop at the anus as the result of venous stasis.

### TREATMENT OF CHRONIC CONSTIPATION

*The treatment of chronic constipation* and its success depends upon the exact recognition of the special cause in the individual case. With the removal of the cause the symptom disappears of itself. The constipation of breast-fed children is readily relieved. With abundant food, its proper assimilation, and the absence of any organic factor, the constipation is relieved after small doses of tea, meat broth, or malt extract. In more severe cases abdominal massage will bring about the desired result. This is performed by the mother or wet-nurse twice daily, morning and evening, when the child's stomach is empty and best after the bath. The movements are those of light stroking with the oiled hand along the course of the colon, succeeded by kneading of the abdomen.

These manipulations, although conducted with care, must nevertheless be somewhat energetic, if the desired effect is to be produced.

Constipation, the result of over- and under-feeding, soon disappears when the diet is regulated, as was set forth in the discussion of these digestive disturbances.

In Hirschsprung's disease purgatives and daily injections of water into the intestine are necessary to bring about an *evacuatio alvi*. In addition to enemata of water Lennander of Upsala advises the employment of electricity, one electrode being introduced into the dilated colon, the other being used to stroke the abdominal wall. Both methods of treatment promise success, for by the evacuation of the large intestine with injections of water as well



as by electricity the tonus of the diseased intestinal area is raised, the dilatation decreased, and normal conditions are finally brought about.

In addition to their therapeutic effect the intestinal washings are also an aid in diagnosis. With these injections, in case we are dealing with a dilated colon, there is a conspicuously rapid off-flow of the water and an enormous receptive capacity of the lower bowel thus enabling the infant to retain several liters of water. This conspicuous phenomenon, which can be explained only by the abnormal width of the large intestine, with the simultaneous presence of marked meteorism and stubborn constipation, at once indicates the diagnosis to the careful observer.

Recovery in dilatation of the colon is not hopeless although in the majority of cases the disease is life-threatening.

For the narrow longitudinal tears of the anus (*fissura ani*) warm sitz-baths, inunctions with indifferent salves, painting with a 2 per cent. silver nitrate solution or salve, cauterization with silver nitrate, painting with a 10 per cent. cocain solution or inunctions with a 5 per cent. cocain salve are of service. Ichthyol suppositories are also valuable:

R/.

Ammon. sulph. ichthyol.....	2.5
Cocain. hydrochlor.....	0.05
Ext. Belladon.....	0.01
Butyr. cacao Unguent. Cerae.....	q. s.
f. suppository.	

I have used the suppository treatment in several cases of anal fissure with extraordinary results (recovery after 8-10 days) and believe it to be a very valuable aid in the cure of this affection.

Division of the sphincter for the purpose of relieving the tension of the lower portion of the bowel and for the relief of pain I have never found necessary but the introduction of a metal rectal bougie of the thickness of the little or middle finger often produces a gradual, moderate distension of the sphincter and relief of the pain.<sup>1</sup>

In the treatment of hemorrhoids sitz-baths and inunctions play an important rôle; with decided nodular formation an ice-bag is of service. Anusol suppositories, which consist chiefly of bismuth, zinc oxid, and balsam of Peru, are of advantage. In both anal fissure and hemorrhoids we must resort to internal medication for a soft pappy stool.

The greatest resistance to treatment is presented by those forms of chronic constipation in which no obvious cause can be demonstrated and in which, therefore, it is necessary to invoke the congenital peculiarity of the infantile intestine and the so-called atony in explanation. Dietetic measures should first be tried. Accordingly such errors of diet as may exist are corrected

<sup>1</sup> Forceful dilatation is the quickest and easiest remedy. A drop of a  $\frac{1}{2}$  p. c. solution of silver nitrate along the fissure afterwards every 3 or 4 days. Cleanliness.—EDITOR.

either by a greater concentration of the milk mixture or by a reduction of the milk and a greater addition of water. Sometimes it is possible to influence the sluggishness of the intestine by the addition of larger quantities of cream or sugar than are customary for a normal child of corresponding age. Sugar of milk is not well suited for this purpose but dextrose and malt sugar are particularly valuable.

To every bottle of milk is added one or two teaspoonfuls of a concentrated solution of dextrose (10-20 per cent.), malt powder (Brunnengräber), or malt extract (Loefland, or the milk regime is interrupted for a few days by the substitution of Liebig's or Keller's malt broth. An increase of fat may be produced by the addition to the milk of one-half to two teaspoonfuls of cream or butter to each bottle. Fresh butter seems to have a more prompt effect than when cooked with the milk. An increase of fat is also brought about by the well-known milk modifications, such as Biedert's cream mixture and Gärtner's fat milk.<sup>1</sup>

Should these dietetic measures prove ineffectual a movement of the bowels may be induced by soap or glycerin suppositories, or by the injection of water, oil, or glycerin. In very obstinate cases the only alternative is to remove the compact fecal masses with a well-oiled rectal bougie or long-handled curette, or with the finger. These methods are only successful for the single movement; to attain a permanent result particularly in the anatomical condition, there is no better remedy than abdominal massage, the technic of which has already been given. If this method is practised energetically and without interruption for four to six weeks the disagreeable condition will in the majority of cases permanently disappear.

At the commencement of this mechanical treatment and occasionally during its course we cannot dispense with the administration of a laxative. In addition to the plentiful administration of cream and the special preparations of sugar the following remedies are useful in infants:

Oleum ricini 5-10 grams, magnesia 3-6 grams, pulv. magn. cum. rhei (Ribke's infant powder) 2-5 grams, pulv. glycyrrhiz. composit. (Kurella's powder) 1/2-1 teaspoonful, infus. sennae composit. (Viennese potion) or vinum rhei in teaspoonful doses, syr. spin. cervinae s. rhamni catharticae, a small teaspoonful, and at the termination of the nursling period pulv. rhei, a knife-tip full, etc., etc.

In conclusion I must call attention to the influence of prophylaxis, or the early and persistent treatment of chronic constipation in infancy, upon the frequency of that condition in advanced childhood and in puberty, which indicates the necessity for employing all practicable measures for the relief of this anomaly in its earliest incipency.<sup>2</sup>

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<sup>1</sup> What about Rotch's methods, or Holt's?—EDITOR.

<sup>2</sup> The muscular incompetency depending on early rachitis and resulting in and after the third month of life has not been mentioned. It is a frequent occurrence, and requires antirachitical treatment.—EDITOR.

## CHRONIC DIGESTIVE DISTURBANCES OF BOTTLE-FED INFANTS

In regard to the prognosis these maladies are in coarse contrast to the various affections of breast-fed infants. A chapter in the pathology of nurslings is here unfolded which, as regards the etiology and pathogenesis, is one of the most obscure in the investigation of diseases of childhood, and as to the results obtained by therapeutics requires the most strenuous efforts of even the experienced pediatricist.

In the chronic digestive disturbances of artificially-fed infants we find all of those anomalies of the fecal movements which we have learned, from the pathology of the acute gastro-intestinal affections of infancy, to recognize as dyspepsia, intestinal catarrh, and enteritis. Here, however, as in the other chronic diseases, the acute, tumultuous onset of the process is lacking and the various forms are not sharply defined, losing their characteristic type and merging into or alternating with one another.

“Simple” chronic intestinal disease of the artificially-fed infant, which occurs in association with an acute affection, should be separated from the “severe” type, the termination of which, under the picture of extreme emaciation, we recognize as atrophy (*pedatrophia* or *athrepsia*, Parrot). This clinical picture, to which the English give the name *marasmus*, was formerly, through a false conception, described under the name *tabes mesenterica*. A sharp distinction of the two pathologic processes is not intended by this division for often enough the gradually declining processes occurring in connection with acute digestive disturbances of nurslings present the typical picture of atrophy. But even more frequently is the complete picture of atrophy as a disease *sui generis* developed without preceding alarming disturbances on the part of the intestine.

## SIMPLE CHRONIC INTESTINAL DISTURBANCES OF ARTIFICIALLY-FED INFANTS

As a rule these originate from an acute intestinal or gastric catarrh. The abrupt symptoms, the fever, and the diarrhea subside in the course of a few days and with this an apparently mild subacute disturbance appears without especially prominent phenomena on the part of the digestive apparatus. In other instances the affection develops more insidiously, with less obvious symptoms. Slightly altered dyspeptic feces of greenish yellow or green appearance, or a gray or grayish yellow and occasionally dry stool, introduces the disease. In other instances there is a more marked involvement of the small intestine, as is evident from the fluid evacuations, or an affection of the colon is indicated by an admixture of mucus, blood and pus. Quite often a varying picture is observed: the dyspeptic and catarrhal symptoms alternate, always, however, in apparently mild form and slight intensity.

Vomiting is now and then noted at the onset, the milk being regurgitated in small or large masses soon after its ingestion. In the further course of



the malady this symptom is more rare, only occasional small, clumpy, coagulated, very acid masses being ejected from the stomach. In many infants symptoms on the part of the stomach are lacking.

Microscopic examination of the feces shows nothing characteristic of chronic catarrh. The chemical analysis of the gastric contents shows in almost all cases a decreased hydrochloric acid secretion.

The constitutional condition of the child appears to be but little disturbed. Fever is commonly absent, but it may appear intercurrently with a lapse of dietetic stringency or from complicating purulent or septic processes.

Colic is uncommon although after the ingestion of food there is at times a decided restlessness. As a rule the abdomen is not distended nor tense but rather pliable; as the affection progresses, however, there is retraction and the abdominal coverings are depleted of fat and become so thin that the internal organs and the mesenteric glands are readily palpable.

The disposition of the child, at first but little influenced, eventually changes to fretfulness and whining. The smiles which are common in health and are such a source of happiness to the mother, become rare and at last disappear altogether from the face of the ailing child. In the course of time the complexion becomes pallid, the expression languid and suffering, although the eyes are bright and clear in contrast to the furrowed and aged features.

The appetite is often poor or entirely lacking; occasionally it appears to be increased since the child drinks eagerly from the bottle, but the food is frequently soon vomited.

The tongue is usually coated; in the further course of the disease it is often glistening red and simultaneously the entire mucous membrane of the mouth is intensely inflamed, sometimes thickly covered with thrush or merely with isolated aphthous loss of substance. Salivation may be increased or lessened. The odor from the mouth is fetid or slightly putrid.

The amount of urine is somewhat decreased and almost always contains sediment and often albumin.

While at first the arrest or even the loss in weight is scarcely obvious, especially if weighing at regular intervals is not practised, in the further course of the disease and perhaps in a short time the emaciation of the entire body becomes apparent and is particularly conspicuous in the face and extremities. The skin and muscles lose their fat, producing loose folds which lend to the entire body a withered appearance and to the face an aged expression. The abdominal skin becomes thin and flaccid and, owing to the loss of its elastic tension, forms into folds.

These symptoms gradually attain greater prominence, the emaciation becomes more conspicuous and impresses its characteristic stamp upon the infant.

The weight sometimes declines slowly, at other times rapidly, but the decline is not necessarily continuous; on the contrary, there is occasionally a period of apparent improvement with a transitory moderate gain in weight, which in time is succeeded by a loss. Thus, according to its gravity, the con-

dition may continue for weeks or months, and even then, with suitable treatment, may terminate in recovery.

In other cases of protracted course the child gradually loses strength and finally perishes with the manifestations of cerebral anemia, cardiac collapse, or in an eclamptic seizure. Convulsions are extremely common in the terminal stage of the affection. A portion of the children succumb to a renewed acute intestinal disturbance—intestinal catarrh, enteritis—or with the toxic symptoms of cholera infantum. The remainder of the patients pass into the lingering invalidism of atrophy.

The most frequent complications of chronic intestinal catarrh in bottle-fed infants are intercurrent acute gastric or intestinal disturbances; now and then severe vomiting. In addition the skin and the mucous membranes, on account of their decreased resistance, provide excellent points for the entrance and colonization of microörganisms of the most various kinds which give rise to diseases of the skin, such as ecthyma and furunculosis, and upon the mucous membrane of the mouth to thrush and other forms of stomatitis. Otitis media is not an uncommon complication. Frequent eclamptic attacks, diseases of the bronchi and of the lungs, inflammatory conditions of the bladder and of the kidney, render the prognosis serious and in many cases endanger life.

Besides the atrophic conditions which we have learned to recognize as the frequent termination of a chronic intestinal catarrh there is a picture of

### ATROPHY

of more typical form and in its highest ascendancy, which we are disposed to regard as *genuine atrophy*, as a disease *sui generis*.

By this genuine atrophy, therefore, we understand a chronic nutritive disturbance of artificially-nourished infants which is characterized by a constantly increasing atrophy of the muscles and of the fatty tissue, and which finally leads to a permanent arrest or continuous loss of weight and to extreme emaciation.

In this consideration of primary atrophy all of those *secondary atrophies* are excluded which are accompaniments of tuberculosis or syphilis or are the consequence of severe acute lesions of the digestive tract and of exhausting diseases.

The so-called "*hunger atrophies*" of breast-fed infants are also omitted which have already been described and which, as a rule, owe their origin to insufficient food from a breast that secretes an inadequate quantity of milk without the knowledge of the inexperienced mother. Nor in the description of this special nutritive disturbance shall we discuss the conditions of hunger and death from starvation, such as are brought about artificially in the so-called baby farms ("*Engelmacherinnen*") by means of hunger cures or the gradual withdrawal of food. We shall limit ourselves to the genuine atrophy which arises with an apparently rational and adequate nourishment and in fact appears mysteriously without gross digestive disturbance, I might almost say without a demonstrable cause.

The development of the affection is very gradual. Children previously in apparent health and developing normally gradually become pallid and feeble. The increase in weight is no longer satisfactory or an arrest has occurred. Very slowly and unsuspectingly the phenomena of emaciation and its consequences become obvious.

The pallor increases, the infant appears completely emaciated; the upper and lower extremities are flaccid and wasted, skin alone covers the chest and back so that every individual rib is outlined. The bones of the skull overlap, the fontanelles are often depressed. The face shows numerous folds, especially upon the forehead; the bones of the face project, the temporal region is depressed, the cheeks are hollow, there are sharp lines about the nose and mouth; the nose stands out prominently. All of these phenomena convey to the infantile countenance the physiognomy of senility. The picture develops with the progressive disappearance of fat and constantly increasing emaciation and loss in weight.

The expression of the child is care-worn, with deeply sunken but clear eyes. The disposition is altered, the patient is fretful and whining. The voice becomes hoarse.

Sleep is restless, often light and disturbed. Providing no inflammatory complication is present the temperature is normal or frequently subnormal.

The digestive function of the little patient in many instances is undisturbed. The appetite is usually good, sometimes even increased; the fluid requirement appears to be very great and even qualitatively the child demands more than the normal infant. It often grasps the bottle hastily and when tormented by hunger empties it rapidly. In the intervals between the feedings the child forces its fist into its mouth, presenting the picture of insatiable greed.

The fecal evacuations, in many instances, are perfectly normal, in others there is an intercurrent, very transitory dyspeptic or catarrhal condition, and more rarely normal periods alternate with mild or severe intestinal disturbance.

With the symptoms above described the child emaciates more and more until it appears almost a skeleton. The adipose tissue and the muscles of the entire body have disappeared until nothing but skin and bones remains. The skin, as a covering for the tissues in and around it, has become too loose and "hangs" from the bones.

Ohlmüller has shown by an analysis of a normal and an atrophic infant of the same age that the decrease of weight in atrophy is to be ascribed principally to the loss of the fatty tissue. In the case examined by this author, in which 57 per cent. of the total weight had disappeared, 90.9 per cent. of the fat supposed to have existed in the body previously and 30.7 per cent. of the albumin substances were lacking. The loss of solid constituents from the heart, the liver, the brain, and the bones was much less than that from the muscles, the skin and the intestines.

The face has the appearance of senility; the skin and the mucous mem-



branes very often reveal inflammatory conditions (aphthous and other forms of stomatitis, intertrigo, ecthyma, furunculosis). The same diseases which arise as complications in the simple, chronic, intestinal disturbances are also common here. Aside from eclamptic and tetanic conditions, which are especially frequent *sub finem* but which may often develop no further than a slight spasmodic muscular tension in the extremities and in the nape of the neck or simply an increased restlessness which is sometimes alternated with apathy, processes of especially purulent and septic nature are noted, as affections of the intestine, lungs, bladder, kidney, and brain, which are either a transitory menace to the infant or are the termination of the tragedy. During the last hours prior to death there is occasionally, as in other severe conditions of intoxication, an irregular, rapid and deep respiration (acid respiration, Czerny).

Under this picture of distressful misery the infant in extreme cases sinks deeper and deeper in consequence of the unabated and disastrous loss of weight, and ultimately perishes with the signs of hyposthenia but without special symptoms of disease.

In regard to the course and termination of "genuine" atrophy there can be no doubt that the prognosis for infants treated in the hospital is more unfavorable than for those of private practice. Of the various points which demonstrate this seemingly contradictory statement it must be remarked that in all probability in hospitals the monotony and errors in nursing are not so much at fault as the possible action of infectious toxins (toxin producers) which have accumulated in the wards of the hospital and in some manner find entrance to the organism, causing an inhibition of the normal development and consequent emaciation. In private practice these influences (hospitalism), of which the latter even in the hospital is operative only periodically and transitorily, are usually absent, and therefore the chance of recovery is very much more favorable. Even in the the hospital, however, a cure of the atrophic condition is possible and in fact is often observed; it is true that at certain times our curative endeavors find a more favorable soil than at others, and from clinical observation the impression is gained that the miasmatic agent which terminates severe marasmus comes and goes in the hospital, or at all events is not permanently present.

In regard to the course of "genuine" atrophy all possible variations may be considered. Apart from the complications that have been mentioned, only the degree of emaciation and the amount of weight lost or the length of time the weight is arrested furnishes an indication as to whether the affection must be regarded as hopeless or whether we are to encourage the parents. But in the decision of these two factors even the experienced physician is frequently in doubt, since, after a hopeless condition of invalidism, an almost unaccountable improvement may be observed just as frequently as a sudden catastrophe after days of apparent betterment.

Thus, cases will be observed which speedily terminate in recovery after brief treatment. In other patients there is a slow gain in weight which in

turn declines and rises again, and after numerous alternations falls rapidly and the affection terminates fatally. Or after several increases with succeeding "catastrophies," as Heubner calls this repeated rise and fall, the resistance of the organism finally gains the victory and the process terminates in recovery. Again, for weeks or months there may be only a slight gain or loss, the weight curve showing an almost straight line, but eventually, through decrease in strength or the appearance of some complication, death takes place, or inversely, by a sudden increase of weight, improvement and complete recovery result.

This peculiar symptom-complex, which is here demonstrated by the weight curves (Figs. 4-10)

offers the greatest difficulty in regard to its etiology. At least it is impossible at the present time to form a clear and complete picture of the processes which

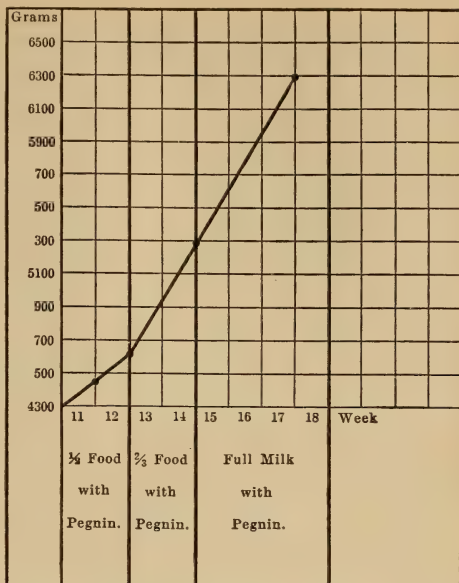


FIG. 4.—P. G., aged 11. Undiluted milk with pegnin. Vomiting and arrest of weight. Diagnosis: ATROPHY.

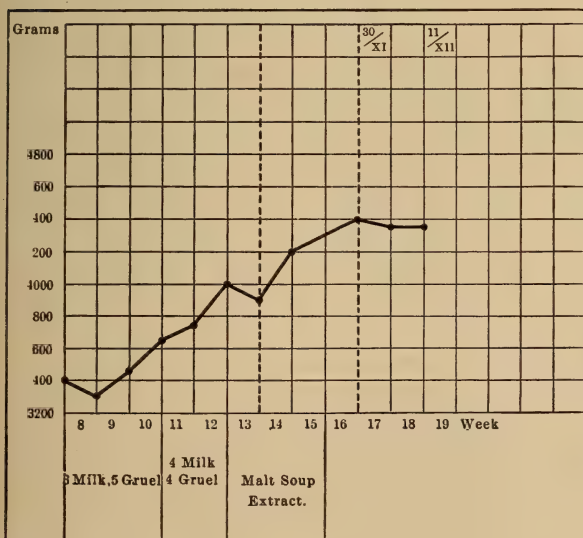


FIG. 5.—E. H., aged eight weeks. Keller's malt soup. Atrophy.

are operative in these chronic, nutritive disturbances in the infantile organism and especially in the metabolism. It must be admitted that in some of the cases chronic digestive debility produced by hypernutrition or improper feeding is a predisposing factor, or that an occasional, transitory, chronic, trivial, digestive disturbance plays an etiologic rôle in the introduction of the process.

It would seem obvious that in a disease running its course with such

a conspicuous loss of fat of the skin, of the muscles, and of the internal organs we should seek the explanation in a severe lesion of the intestine, as the secretory and resorptive apparatus. As a matter of fact this theory has been invoked by some investigators to explain this peculiar chronic anomaly.

The main exponent of this view is Baginsky, who even to-day maintains that "atrophy is the consequence of disturbed assimilation arising from atrophic alteration of the intestine." In support of this view he offers two important points; first, the severe atrophic and degenerative anatomical lesion of the intestinal tract as regards its epithelium and its glands, and second,

the enormously decreased resorption of the important constituents of the food.

In the post-mortem examination of the mucous membrane of the intestine of infants who had succumbed to atrophy Baginsky found various conditions ranging from a primary inflammatory irritation with infiltration of the mucosa to chronic inflammatory swelling and proliferation of the villi, glandular structures, and follicles. These severe changes of hyperplastic nature in the glands and villi are succeeded by desquamation of the proliferated and loosened tissue and finally by a destruction and atrophy of the surface of the intestine.

Although the enigma of atrophy in infants is best solved upon the basis of an anatomical lesion

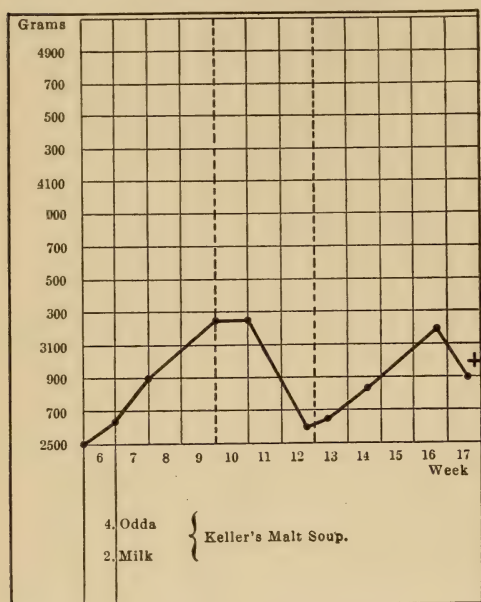


FIG. 6.—A. R., aged six weeks, a twin. Atrophy.

there are so many considerations based on other post-mortem findings (Heubner, Habel, Finkelstein, Fehde, Bloch, E. Meyer) which are opposed to this view of Baginsky's that we cannot agree with him. A number of cases of unquestionably genuine atrophy are reported in which minute investigation of the contracted intestine has shown that all the glands and villi are well formed, Lieberkühn's follicles pressed close together and covered with epithelium, and which exclude any gross lesion of the intestinal mucous membrane and of the remaining intestinal wall in all of its parts. The lesions described by some authors as those of intestinal atrophy may be produced by inflation in any normal bowel, in fresh as well as in decomposed intestines. So-called spontaneous intestinal atrophy has never been found in contracted intestines. Therefore we must conclude that the lesions which are supposed to indicate atrophy may be referred to an altered physical condition of the intestinal wall; in this case



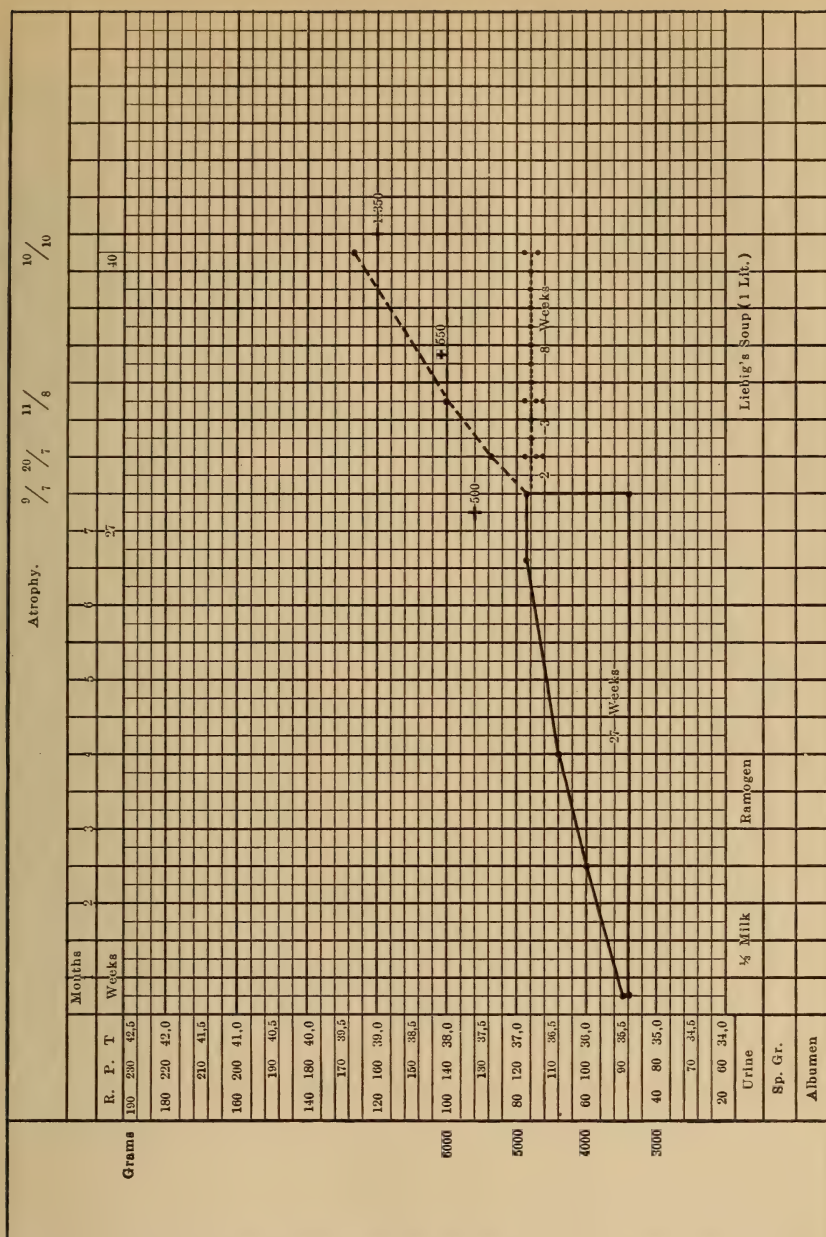


FIG. 7.—F. D., born December 9, 1902. Weight 3600 grams. Brought for treatment July 7, 1903, "because it does not gain in weight." Constipation; cries much; occasional vomiting. Food, one-third milk, later ramogen. Never developed properly. Treatment with peginin added to one-third milk.

Weight at beginning of treatment 3600 grams  
 at twenty-seven weeks 4800 " —normal 7500 grams  
 Gain 1200 "  
 Gain per week 40 "

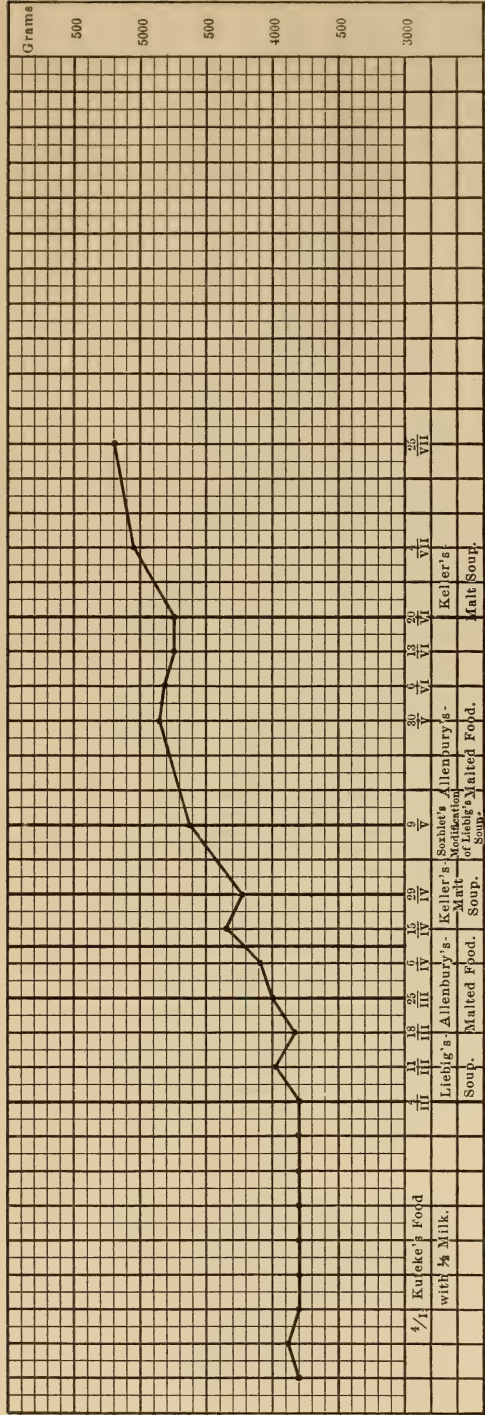


Fig. 8.—Köhler, two and one-half months. Atrophy.

the anatomical proof of a true atrophy of the mucous membrane in general atrophy of nurslings cannot be demonstrated and the entire theory founded upon this teaching is untenable (Heubner). However, I do not pretend to doubt the fact that, like the other tissues, the intestine of the shrivelled child is atrophied, i. e., emaciated, as has been proven by Ohlmüller's investigations previously quoted.

A severe anatomical lesion of the mucous membrane of the intestine would not coincide with the clinical facts, since we have seen from the course of the affection that occasionally after weeks of invalidism the entire picture suddenly changes, improvement takes place, and finally recovery.

In a severe anatomical defect of the mucous membrane repair in so brief a time cannot be considered. Baginsky explains these sudden improvements by the fact that in addition to the more or less altered or completely destroyed areas of the intestine there are also quite normal portions which are intact and covered with well-retained epithelium.

In spite of the lack of proof of atrophy of absorbing intestinal epithelium it cannot be denied that in some cases, in so far as assimilative investigations in atrophic infants are at hand, the power of absorption appears to have

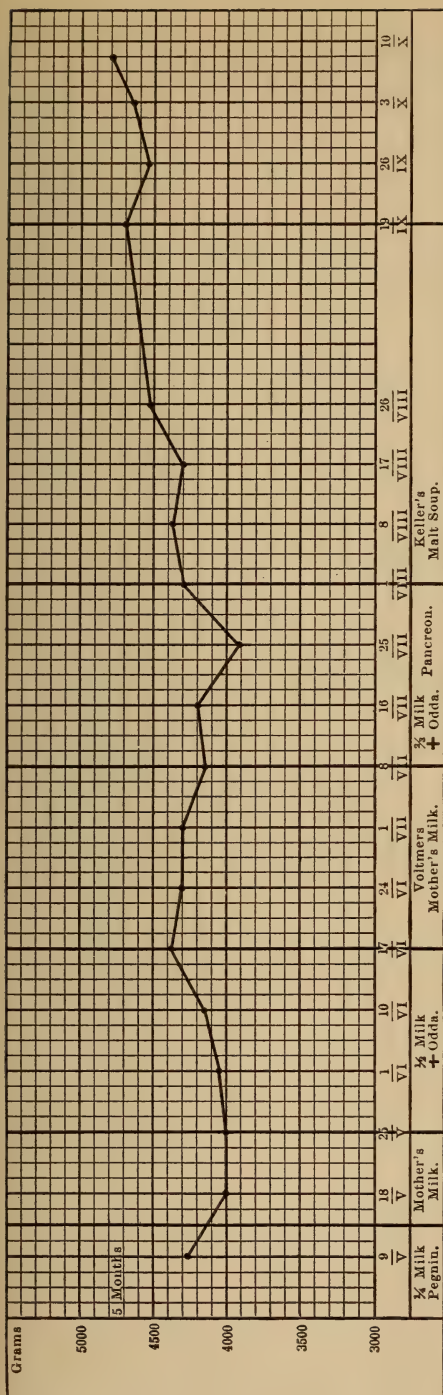


Fig. 9.—W. B., five months. Atrophy.

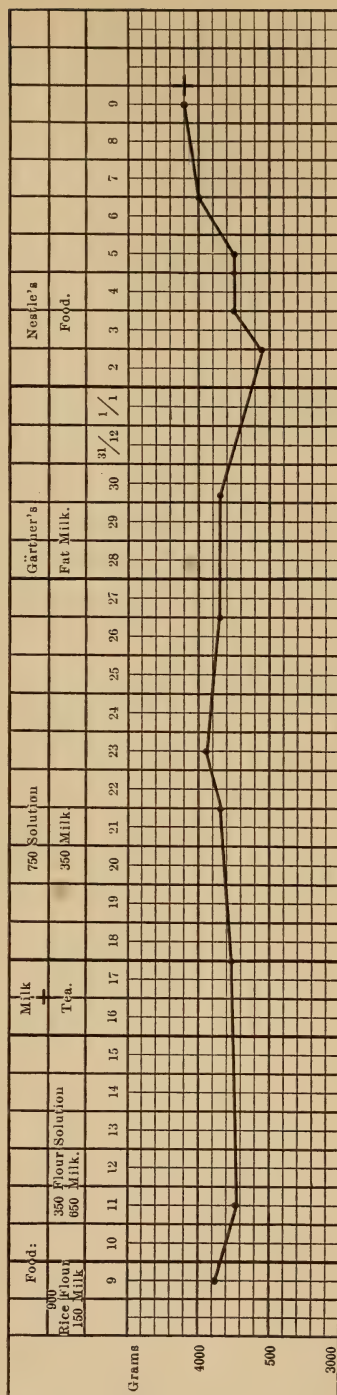


Fig. 10.—J. P., four months. Atrophy.



suffered. Baginsky found, in three cases where the intake and output of the N-containing bodies had been determined, that in the first case the N-loss was 52.7 per cent., in the second 37 per cent., and in the third 45.2 per cent., in contrast to the normal N-absorption which, according to the investigations of Förster, Uffelmann, Camerer, Lange, and Bendix, amount to 80-96 per cent.

Heubner also saw two cases of genuine atrophy in infants with skeleton-like emaciation in which the absorptive power of the intestine was so decreased that in one instance 43.7 per cent. and in the other 54 per cent. of the introduced nitrogen was unabsorbed.

From my own investigations, with the exception of the case observed with Heubner, it appears that with a rational feeding a decrease of nitrogen assimilation does not exist in the atrophic child to any marked extent, as I have found values for the absorption of nitrogen of 71.96 to 85 per cent.

In a review of the literature regarding metabolism of atrophic nurslings the figures for the resorption of nitrogen are 86-95 per cent. (Freund) and 81-95 per cent. (Keller); Lange also reports similar figures.

In contrast to this slight loss in nitrogen (except in Baginsky's and Heubner's cases) I was enabled to determine *one* fact in my investigations of the metabolism in atrophic infants, namely, a decided reduction in the absorption of fat. In some instances the loss amounted to 40-50 per cent.

Although the decreased power of absorption certainly plays an important part in the deficient gain in weight or in the progressive deterioration of the health of the atrophic infant this does not entirely explain the resulting atrophy, as in some instances, with a fair intake of nitrogen and in spite of greatly reduced fat absorption, sufficient heat units are taken up and even more than are necessary, according to our present views, for the energy requirement of the infant—for the functional activities as well as for an increase in weight.

At the present time we calculate the "energy quotient" = the number of heat units for any given weight of the body, as 100 calories per kilo per day for the normal infant. In one of my experiments an atrophic child was given 125 calories per day and per kilo. If, however, in addition to the decreased absorption of food there is an increased expenditure of energy, which is principally lost through digestive function and by destruction of the cells, as may be recognized by an abnormal increase, in the interchange of gases, there would be in these two components together a better explanation for the atrophic condition.

Heubner has illustrated the disproportion in the atrophic infant between the intake of energy and the energy loss by a simple equation with the symbols first utilized by Camerer. If  $n$  = energy of the food,  $e$  = energy of heat radiation and of evaporated water,  $l$  = energy of substance given off from the body,  $v$  = loss in absorption, it is clear that when  $e + l + v$  becomes greater than  $n$ ,  $a$  becomes negative, i. e., loss in weight must take place.

With an appreciation of these relations of metabolism it would be clear

why the weight of the child is arrested or even diminished, but it does not explain how the atrophic infant acquires this peculiar debility of digestion and why, in the metabolism of its nutritive material especially, a far greater labor is required than under normal circumstances. In addition there must be explained the accompanying phenomena of atrophy of the skin, the mucous membrane, the kidneys, and the nervous system, which have already been explicitly detailed.

In explanation of such changes it has been thought that these complications, like the deleterious atrophic conditions in general, may be referred to intoxications which have arisen from the destruction produced in the intestinal tract by saprophytic or specific bacteria.

In addition to chronic poisoning by infectious toxins a disorganization of the constructive material, as Finkelstein believes, may also arise from auto-intoxication, since, either in the intestine or in the intermediary metabolism, deleterious products of a non-infectious variety are formed or substances which are normally unabsorbable are assimilated. To the present time all attempts to demonstrate such toxins have failed; neither from the contents of the intestine nor from the intestinal wall has it been possible, at least in the animal experiment, to obtain substances which could be proven to be toxic, either by feeding, by intraperitoneal injection or by inoculation into the blood current.

As an explanation of the atrophy of infants Czerny has given a definite form and conception to the idea of an intoxication of the organism. He regards this disease as a chronic nutritive disturbance produced by an acid intoxication from hyperacidity of the organism (acidosis) which necessitates a greater activity, to which it is unequal on account of the toxic effect of the circulating acids. According to Czerny the details of this acid intoxication are as follows: Through improper feeding and especially through a surfeit of food, acids arise in the decomposition of albumin products and in the splitting of fats, which act upon the organism as poisons even when no toxins or harmful bacteria are introduced into the intestinal tract. In this process milk sugar is of less consideration because of its high assimilative limits. Czerny concludes, however, that the acid intoxication not only has the importance for the organism of making nourishment difficult or even impossible, but that also by the continued decrease of alkalinity an essential part of those properties is lost which protect the organism from infection. In the former condition a food which contains normal amounts of albumin and fat, well borne by healthy children, produces threatening symptoms of intoxication; a deficit of albumin necessitates the consumption of the child's body albumin and causes a rapid decline in weight. In the latter case, infants suffering from acid intoxication may develop secondary infections from the intestine and in consequence the clinical pictures which at first are quite similar, may in their further development be extremely diverse. Thus there is a permanent vicious circle dependent upon the intake of food and, resulting from this, acid intoxication, insufficiency of function, and disturbed nutrition.

This theory, propounded by Czerny in 1897 upon the basis of clinical facts, and at that time but little credited, has by the incessant labor and painstaking investigation of this author and his pupils (Keller, Freund, Thiemich, v. d. Berghs, Steinitz) developed from a firm foundation into a stately structure, but does not furnish an absolutely reliable explanation for all cases and under all circumstances. The actual outcome of Czerny's theory of acid intoxication is Keller's important finding of an increased excretion of ammonia through the kidneys in 10 out of 11 infants with chronic intestinal disease. Because of the amount of ammonia found in normal adults and of the findings in the healthy new-born, the Breslau school came to the conclusion that the absolute and relative increase of ammonia observed in the urine of infants with chronic gastro-intestinal disease represented a disturbance of the intermediary metabolism. An explanation of the increased excretion of ammonia when acids circulate in the body is given by the following chemical process: If there is an excess of acid in the organism and the quantity of fixed alkali present is insufficient for its neutralization, ammonia takes its place, is absorbed by the acids for the formation of salts for excretion, and is passed with the urine even before its further reduction into salts of ammonia. The excretion of ammonia is relatively greater, that of urea less, than normal.

That the increased excretion of ammonia in the urine, determined by Keller, does not depend upon a disturbance of the hepatic function which produces urea but upon an excessive production or decreased oxidation of acid products of metabolism is obvious from the investigations of v. d. Berghs, according to which, after the administration of alkalies (Schröder-Münzer's criterion) a combination of acids, a transformation of ammonia into urea and its excretion as such, takes place as under normal conditions.

As fascinating as is Czerny's theory of acid autointoxication, nevertheless quite a number of links in the chain of evidence are lacking which we are accustomed to require in the well-known adult types of acid intoxication (diabetes, carcinoma, leukemia, etc.) and which have been demonstrated in these maladies. Among them are the decrease in the alkalinity of the blood, increased acidity of the urine, with the demonstration of physiologic acids in increased amounts or of pathologic acids of various kinds in the urine.

After the failure to demonstrate these points it must be assumed, as Pfaundler quite properly asserts, that in chronic gastro-intestinal disease of nurslings, if an acid autointoxication is present at all it cannot be such as is ordinarily implied and with which we are familiar in the pathology of adults.

To these deviations from our ordinary conception of acid autointoxication a few factors must be added which are calculated to raise a doubt as to whether the explanations of the Czerny school for the chronic nutritive disturbances of infancy are conclusive or absolutely reliable. It has been shown that high ammonia values are occasionally present in normal nurslings (Pfaundler) and it is also positive that they are not invariably present in nurslings with



chronic intestinal affection—a fact which I was the first to prove by positive investigation, and which was later confirmed by Pfaundler.

A further especially important fact which I demonstrated in my experiments of 1898 is that the co-efficient of ammonia—as Pfaundler terms the percentage of nitrogen excreted in the form of ammonia, compared with the total amount of nitrogen—depends upon the quantity of fat in the food. This fact is confirmed by Keller's findings, which prove that the high excretion of ammonia is made greater by the administration of milk rich in fat, or of cream, but which falls to normal as soon as milk containing but a moderate amount of cream or even deficient in cream is administered.

In view of these relations the question of autointoxication appears in quite a different light. Strictly speaking, we should not even refer to an autointoxication, as Pfaundler has declared in a recent critical review of this question, as we are dealing with an external effect through the composition of the food, which does not present a demonstrable deleterious influence as the immediate consequence, but, on the contrary, an "alimentary hyper-acidity" or a "fat-feeding acidosis" (Pfaundler).

As, however, infants with normal digestive function react to an increased administration of fat in the same manner as patients with gastro-intestinal disease—according to Pfaundler, by an increase in the ammonia co-efficient—it cannot be said that an increased excretion of ammonia in the urine is a positive criterion of a chronic nutritive disturbance of nurslings.

In addition to the high amount of fat in the food of infants, which produces an alimentary acidosis in both the healthy and sick, the high renal excretion of ammonia may also be due in part to a certain lack of oxidating activity, or, in children suffering from chronic gastro-intestinal disease, even a degeneration of the hepatic tissue may be held responsible (Pfaundler, Brüning).

Therefore, after all the theories that have been invoked to explain the chronic digestive disturbances of infancy, the symptom-complex which we have designated atrophy or athrepsia still remains unsolved. However, that atrophy is in relationship with and dependent upon the nature of the nourishment may be regarded as certain since it accords with experimental findings and clinical facts. That improper food is not infrequently the starting-point of this serious disturbance is undoubted, and just as true is the observation that by the administration of "correct" food the severely damaged general condition may be improved and the affection brought to a favorable termination.

## TREATMENT OF CHRONIC DIGESTIVE DISTURBANCES

In the *treatment of the chronic digestive disturbances* of infancy it will be necessary, in evolving a plan of cure, to differentiate between the "simple" chronic affections, which arise in conjunction with acute maladies, with chronic dyspeptic or catarrhal fecal discharges and simultaneous feebleness and emaciation of the infant, and "genuine" atrophy.

For both forms the general rule may be advanced that the best and most curative agent is breast-milk wherever there is a possibility of its administration. In carrying out this plan of treatment, however, insurmountable difficulties may arise. The child who has already cut its teeth bites the nipple while nursing, the breast becomes sore, and this injury and the resulting pain necessitate weaning. In other instances the debility of the infant prevents the withdrawal of the required quantity of milk. The aid of the milk-pump for this purpose fails because the amount of milk obtained is too small and the gland is not emptied sufficiently, in consequence of which a stasis arises which in itself affects the quality of the milk and gradually leads to an arrest of the secretion. Therefore, while in all cases of intestinal disturbance which arise in artificial nutrition it is advantageous to advise natural food, nevertheless, on account of the difficulties that have been mentioned the attempts in this direction will in some instances be fruitless.

If natural food for one or another reason is impracticable, artificial nourishment must necessarily be employed. In adopting this plan I believe it to be important, as in the treatment of acute digestive disturbances, to cleanse the digestive tract and to institute a hunger or rest diet for the intestine to determine the measure of the intestinal secretory and absorptive activity after this period of rest. Therefore, in treating an infant with chronic intestinal disease or with atrophy, the following is the method of procedure:

A single *gastric lavage*, repeated perhaps if there be continued vomiting. The evacuation of the stomach contents may at the same time serve for estimating its acidity. A *mild laxative* to empty the intestine; I prefer four doses of 0.03 grams each of calomel at intervals of three hours. The effect of the remedy is shown by a green discoloration of the feces. Instead of this a teaspoonful of castor oil may be given three or four times during the day. After the lavage of the stomach food should be withheld for five or six hours. This is followed by the so-called *hunger cure*. For this purpose we utilize either dilute decoctions of tea with the addition of sugar or saccharin, or simply boiled sweetened water, perhaps with the addition of ten to twenty drops of brandy to every bottle when debility is present. This hunger cure should be continued for at least twelve hours. The intervals between the individual feedings should amount to at least three and one-half hours; the quantity may be somewhat larger than under normal circumstances (150-200 grams). Upon the hunger days the stool is scant, and with a previous catarrhal or dyspeptic condition it may be regarded as a favorable sign of the activity of the intestine if the fecal mass is formed.

This hunger diet is to be succeeded by a period of rest or forbearance for the bowel amounting to forty-eight hours. For this purpose the child is given a decoction of the "simple" starches. By these we understand barley, oats, wheat flour, mondamine, arrow-root, tapioca, etc., not the manufactured or milk flours which consist of a mixture of milk with some form of flour and in which the starch by some process has been changed into



dextrin. These flour mixtures are to be quite thick, 8-10 per cent.: A tablespoonful and a half of flour is mixed with  $1/2$  liter of water, to which 1  $1/2$  teaspoonfuls of sugar are added while the mixture is constantly stirred. After boiling for fifteen minutes the quantity for one feeding is ready.

If, after this rest-diet, there is a homogeneous grayish-yellow starchy stool, free of mucus and other abnormal constituents, instead of the greenish, chopped, thin or mucus stool, we cautiously proceed to milk food by gradually reducing the quantity of starch and increasing the quantity of milk, its amount and composition to be governed by the age and weight of the child, and its administration to be at first in small amounts and at long intervals. It is best to begin with  $1/3$ -food (350 grams milk, 650 grams mucilaginous material, 52 grams sugar), gradually increasing to  $1/2$ -food (500 grams mucilaginous material, 50 grams sugar), and finally employing more concentrated solutions. In addition to observing the activity of the intestine the nutritive requirement of the infant must constantly be kept in mind. If we remember that the energy quotient of the normal nursling equals 100 we will often note that an infant with chronic intestinal disease, especially at the beginning of the treatment, often requires a much greater supply of food (120, 150 or even 170 calories) to gain in weight.

If the abnormal fecal discharges of infants suffering from chronic dyspepsia do not disappear after a course of the rest-diet and when the milk nourishment has been carefully conducted, in my opinion an infant food might be employed with advantage. Which of these is to be given, whether Nestle's, Neave's, Rademann's, Rademacher's, Theinhart's, Kufeke's, is quite immaterial, as they are all manufactured on the principle that in their chemical composition they should as closely as possible resemble breast-milk.

These infant foods are usually administered in a 5 per cent. decoction, 200 to 250 calories being given per liter.

If improvement of the condition is not brought about by these foods then all of the milk substitutes are to be tried with which modern chemistry of the last decades has supplied us.

The most important and useful of these, from which good results are sometimes obtained, may be arranged, according to definite principles, in the following order (compare B. Bendix, *Milk Substitutes*, in *Eulenberg's Encyclopedic Year-books*, IX, 1900, p. 249):

**GROUP I: MILK WITH DECREASED AMOUNT OF ALBUMIN, ADDITION OF FAT.**

1. Biedert's cream mixture (natural, artificial)—see ramogen.
2. Gärtner's fat-milk.
3. Lahmann's vegetable milk (principle of a cream mixture with vegetable albumin and fat in addition to the milk).
4. Condensed (Swiss) milk.

**GROUP II: DILUTED MILK ENRICHED WITH FAT, THE ALBUMIN OF WHICH IS PREDIGESTED AND MORE OR LESS IN SOLUTION.**

1. Backhaus' milk (addition of cream, solution of part of the casein by trypsin).



2. Voltmer's mother's milk (conversion of cow's milk by the addition of pancreas ferment into pepton; otherwise the composition analogous to human milk).
3. v. Dungern's lab-milk (addition of a pinch of "pegnin" to 200 grams of undiluted milk).

GROUP III: DECREASE OF THE AMOUNT OF ALBUMIN BY DILUTION; INCREASE OF FAT; SOLUBLE ALBUMINATES OF PEPTONS AS A SUBSTITUTE FOR THE DEFICIT IN ALBUMIN.

1. Rieth's albumose milk (substitution of the casein by a non-coagulable albumose prepared from egg albumin by heating); addition of cream and sugar. Similar to Hartmann's somatose milk.
2. Hampel-Lehmann's milk (dilution of cow's milk so that the casein amounts to 0.75 per cent., addition of the yolk of one egg (phosphorus + iron) and the white of one egg. Enrichment with fat and the addition of milk sugar produce a nutritive mixture which in its quantitative composition resembles human milk).

GROUP IV: MIXTURES DEFICIENT IN FAT BUT RICH IN SUGAR (ESPECIALLY MALT SUGAR).

1. Liebig's soup (diastasic action of the flour from malt).
2. Keller's malt soup (50 grams wheat flour + 650 water, 100 grams Löflund's malt-soup extract + 350 milk).
3. Allenbury's infant food No. 111 (malTED food).
4. Liebe's neutral food (Dresden).
5. Soxhlet's nutritive sugar.
6. Brunnengräber's malt powder (Rostock).

GROUP V: BUTTERMILK.

GROUP VI:

- v. Mering's "Odda." Carbohydrate dissolved in part by diastasic action and opened by the baking process, mineral products consisting of  $\frac{1}{2}$  phosphoric acid and  $\frac{1}{4}$  calcium oxide, instead of the fat of butter, the fat of an egg-yolk with lecithin and vitellin, and cacao butter.

Although, as may be noted from their composition, in these substitutes a definite principle has always been maintained, so that by the addition of ferments the labor of digestion is facilitated or some nutritive product is decreased in favor of another, nevertheless it is quite difficult to establish a strict indication as to which disease or under what circumstances any of these nutritive remedies is applicable. Only in two conditions does practical experience serve as an indication. If there be a decrease of hydrochloric acid secretion and if the infant with chronic gastro-intestinal disturbance suffers from frequent vomiting excellent results are often observed from Backhaus's milk, albumose milk, buttermilk, or Dungern's lab-milk. In administering

these modifications, therefore when introducing either predigested or easily absorbed albumin into the food, or perhaps in employing ferments, we facilitate the digestive labor and stimulate the epithelium to a greater activity, wherewith in fact the proper indication is met.

In a second series of cases, those with chronic fatty diarrhea, we also know which substitutes are most useful. Practical experience teaches that here only mixtures deficient in fat should be employed; accordingly Backhaus's milk No. 1, skimmed milk, and buttermilk should be used. In addition infant foods may be given.

In the majority of instances, however, we have practically no indication as to which of the substitutes are to be employed. Often enough the various foods must be tested successively until one has been found which causes the intestinal disturbance to cease and the child to gain weight. One point, however, may be definitely maintained from clinical experience at the bedside: Mixtures rich in fat, such as Biedert's ramogen and Gärtner's fat-milk may be used only with the greatest caution in chronic intestinal disturbances; inversely, nutritive solutions deficient in fat, such as Liebig's soup, Keller's malt soup and buttermilk very frequently bring about good results. Particularly in genuine atrophy do the alkaline soups, deficient in fat but rich in malt sugar, occupy a prominent place in therapy. Among these we include:

#### 1. LIEBIG'S SOUP.

This is prepared as follows: One hundred grams of wheat flour are mixed with one liter of milk, which is stirred in gradually to prevent lumping. This mixture is kept three or four minutes at the boiling-point and is then removed from the fire. Into another vessel 100 grams of malt flour are carefully mixed with  $3\frac{1}{2}$  grams of an 11 per cent. solution of potassium carbonate and 200 grams of water are added. This is mixed with the first solution, the stirring being kept up constantly. The complete mixture is then set aside in a vessel of hot water for half an hour to make it thinner and sweeter, after which it is again placed over the fire and heated until the soup begins to thicken, then removed and stirred for five minutes, and again heated and removed. This process is to be repeated until the mixture is of a thin, fluid consistence and sweet. Finally the soup is boiled for a few minutes and poured through a fine sieve, after which it is ready for use. Liebig's original plan was to prepare each feeding separately; the recipe here given is for the entire day.

#### 2. KELLER'S MALT SOUP.

This is a modification of Liebig's soup and is more easily prepared:

Fifty grams of wheat flour are stirred into  $\frac{1}{3}$  liter of milk and the mixture thoroughly sieved. In another vessel 100 grams of malt soup extract, or 100 grams of malt extract, added to 10 c.c. of an 11 per cent. solution of potassium carbonate in  $\frac{2}{3}$  of a liter of water is dissolved at a temperature of  $50^{\circ}$  C. This malt extract solution is then added to the milk solution and the

entire mixture is boiled. According to Keller Löflund's malt soup extract should be used in the preparation of the food. Keller's malt soup may be used for weeks and even for months as a diet for atrophic infants and sometimes produces a sudden change for the better. In other cases the result is gradual, and at times this method of nutrition is without success.

The chemical composition of the malt soup is as follows: fat 1.0-1.2 per cent., albumin 2.0-2.5 per cent., and sugar 70-80 per cent. The nutritive value of a liter, according to Rubner's combustion experiments amounts to 808 calories. In cases in which the malt soup is without value I have employed a mixture deficient in fat but rich in sugar, and in the preparation of malt soup other malt preparations have been used. Among these are the commercial powders in dry or crystalline form:

a) Liebe's neutral malt extract, to be used in the preparation of malt soup in the same manner as Löflund's malt soup extract.

b) Liebe's neutral food: One-sixteenth kgrm. (four heaping tablespoonfuls) of malt extract mixed with  $\frac{1}{6}$  liter of milk and  $\frac{1}{3}$  liter of water, brought to the boiling point once and the solution poured through a sieve. The soup is then ready for use.

c) Brunnengraber's crystallized malt extract: One hundred grams mixed in a liter of  $\frac{1}{3}$  milk (350 grams milk, 650 grams water) and stirred while boiling.

d) Allenbury's infant food No. III (malted food): Three tablespoonfuls of malted food mixed with  $\frac{1}{2}$  liter of water and  $\frac{1}{2}$  liter of milk, briskly stirred while boiling. After cooling the food is ready for use.

If no beneficial results are obtained even after the employment of these substitutes for Liebig's original malt soup, a food first advised by Dutch physicians (Ballot, de Jager, Teixeira de Mattos) as a general food, sometimes acts admirably in atrophy:

### 3. BUTTERMILK.

The preparation of this food is as follows: Fifteen grams of Kaiser flour are mixed with a few tablespoonfuls of cold buttermilk, added to the remaining buttermilk (one liter) and, with the addition of 60 grams of sugar (and perhaps one teaspoonful of fresh butter), heated for fifteen to twenty minutes and constantly stirred, before the mixture is brought to the boiling point. The milk is allowed to boil up twice and is then emptied, hot, into previously sterilized bottles, sealed with a rubber stopper, and kept cool.

There is great difficulty in obtaining good buttermilk. It must be prepared from sour cream and obtained fresh after the churning. It contains 2.5-2.7 per cent. albumin, 0.5-1 per cent. fat and 3.0-3.5 per cent. sugar. Its acidity amounts to 7 c. c.  $\frac{1}{4}$  n NaOH. The nutritive value is very high: 714 calories to the liter.

Buttermilk is exceedingly well borne by many atrophic infants and in quite a number of instances leads to a permanent reparation of the nutritive disturbance.

I must still emphasize that the trial of a nutritive mixture is not to be



abandoned too soon, and this is of importance in regard to all the substitutes for milk. The organism must become accustomed to any form of nourishment and not before eight or fourteen days can it be determined whether the food is proper or not.

In the simple chronic digestive disturbances in which it is necessary to raise the nutritive condition as well as to ameliorate the chronic catarrhal phenomena and likewise the intercurrent intestinal disturbances of genuine atrophy we must invoke the entire arsenal of *drug therapy* which we employ in the acute digestive maladies.

Among the astringents are tannigen, tannalbin, tannoform (0.5-1.0 every three hours or three times daily), honthine (0.25-0.5), tannopin (0.3-0.5), resorcin (0.1-2.0), perhaps in combination with calomel (0.002-0.003). These remedies must not be abandoned abruptly; in the course of a few days they usually cause constipation and if the remedy is stopped diarrhea is apt to recur. Therefore the drug should be continued for a time in gradually diminishing doses.

If these drugs prove ineffectual, among the metallic agents lead acetate and silver nitrate are of special value:

℞

Plumb. acet.....0.005-0.05

Op. pulv.....0.001

M. f. pulv. S. One powder 4 times daily.

Or

℞

Solut. Arg. nitr..... 0.1

Aq. dest.....50.0

Tinct. Opii gtt. iv-vi or codein.... 0.02

M. S., One teaspoonful every 2 hours.

Argil is also occasionally successful (Soltmann):

℞

Argil depurat..... 1.0-2.0

Aq. dest.....50

Syr. Cinnamomi.....15.0

Tinct. Opii gtt.....iv

M. S., Bottle to be well shaken, one teaspoonful every 2 hours.

Colombo or Ligu. campechiani may be employed:

℞

Decoct. rad. colombo.....10-15.0:100.0

Or

℞

Decoct. lig. Campechiani..10-15:100.0

with or without the additions of tinct. opii gtt. 1-4.

Bismuth, subnitr. or tanicum 0.1-1.25 given in powder form every 2 hours is also serviceable. Neumann advises protargol 0.05.-0.1:50.0 in teaspoonful doses.

*Enteroclysis* is of value in the chronic diseases of the colon. After a luke-warm enema of water the infant is placed in the knee-elbow position and a nozzle the length of a finger is placed on the rectal tube which is also supplied with a funnel. The irrigating fluids to be employed are common salt solution 0.6 per cent., starch enemata with opium, or various other solutions (salicylic acid 0.5-1:500 or tannin 0.5-1 per cent., albargin 0.4:250, plumb. acet. or alumin acet. 2.5:1000, arg. nitr. 1-2:3000). These are often of advantage, but they are also frequently without effect. The quantity for an injection is 50-100 c. c.

In conclusion, in a review of the entire subject of the chronic digestive disturbances of infancy it must be obvious that we are dealing with one of the most difficult problems in the entire realm of pathology. In all of our efforts to conquer this angel of death of early infancy we must aim toward the utilization of two important factors, first the results and application of rich, practical experience in diseases of infants, and secondly a consideration of the individuality of the patient which will exclude a routine method of treatment. Even with the possession and with a comprehensive employment of these factors we have a problem confronting us the solution of which is often beyond our skill.

## INFANTILE SCURVY (BARLOW'S DISEASE)

By H. NEUMANN, BERLIN

At the beginning of the twentieth century we encounter a peculiar disease: of recent recognition and understood by few, its innermost nature not sufficiently clear but probably a disease which will soon disappear, the attention which it has elicited is quite justified. The first clinical and anatomical presentation of the pathologic picture we owe to Möller (1859, 1862), who spoke of the malady as acute rickets, and held that the similarity to scurvy was "purely external and symptomatic." Later the affection was designated "hemorrhagic periostitis," "multiple osteitis," "myelo-periostitis," etc. Its relation, however, to rickets has in the main been occasionally disputed and is to-day upheld by many authors. If we disregard the communications of Ingerslev (1871) and of Jalland (1873) the second period in the history of the disease began with a communication by Cheadle (1878), who demonstrated by its successful treatment the scorbutic nature of the affection. In 1883 Thomas Barlow acceded to this view and amplified our knowledge of the disease by clinical histories and autopsy reports. It appears, therefore, to be another instance of historical injustice that this affection is named after the last-mentioned author. In Anglo-American literature the term applied to the malady is infantile scurvy; in the French it is *scorbut infantile*. I should prefer the designation infantile scurvy. According to the latest investigations this properly characterizes the disease, especially as its relation to rachitis has been found to be quite accidental. It is true we must admit that this is a variety of scorbutus, brought about by external circumstances entirely different from those which prevail in the endemic affection, therefore we shall entirely disregard the designation "Barlow's disease."

In the clinical description of infantile scurvy I shall adhere largely to the newer German experiences; these give slight, transient, and local deviations to the clinical picture for which we will find an explanation later. *The disease begins commonly between the sixth and twelfth months*, rarely in the fifth month, but occasionally between the thirteenth and fifteenth months; older children are not exempt. [Nor are babies very much younger. —EDITOR.] In the summary of 54 observations of Heubner's<sup>1</sup> and in 40 cases of my own scurvy began once in the fifth month, 9 times in the sixth month, 10 times in the seventh month, 20 times in the eighth month, 16 times in the

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<sup>1</sup> Heubner, "Ueber die Barlow'sche Krankheit." *Berl. klin. Wochenschr.*, 1903, Nr. 3.



ninth month, 13 times in the tenth month, 10 times in the eleventh month, 7 times in the twelfth month, twice in the thirteenth month, 4 times in the fourteenth month, and once each in the fifteenth and twenty-first months.<sup>1</sup> In regard to *sex*, in Heubner's and my cases males were somewhat more frequently affected (of 105 children 62 were boys), while in the large American collection males and females appeared to be attacked almost equally. This may be explained by the fact that in the German cases more children developed the disease during the first year—the typical period—when the male sex, as is well known, shows a greater morbidity. The malady usually attacks children who are comparatively healthy and well-nourished and who are not at all or only slightly affected with rickets. Heubner noted severe rachitis only twice in 19 cases, and in 34 scorbutic patients of my own there were 6 mild and 2 marked forms of the affection.

According to early reports infantile scurvy is a disease of the winter and spring months, and is much less intense during the warm season. As a matter of fact there is a conspicuous, although inconstant variation in different months—in 65 cases of Heubner's 36 occurred between March and June, 15 from October to December; in 32 of my cases 14 occurred between March and May and 12 from July to September, the latter therefore during the warm season.

### SYMPTOMS

Prior to the appearance of the disease the child often becomes pale; the ingestion of food may be interrupted to such an extent that the body weight may lessen instead of increase. Usually the attack begins suddenly with tenderness of one leg and early involvement of the other. At first the limbs may be crossed, the normal leg supporting the affected member; later both legs are lightly flexed at the hip and knee and turned outward, or less frequently are drawn up against the trunk. Although this attitude suggests paralysis such a suspicion may be dismissed at once; the muscles are relaxed merely to diminish the pain. Painfulness is evident when the child is dried or bathed, when it is raised or is carried with hanging legs, or when the limbs are touched. Even if the bath has a soothing effect the legs are not moved, and the child cannot be seated because of pain. At first the patient is quiet so long as the limbs are undisturbed; later, however, anticipated movements will provoke crying: raising of the bed-clothes, or the approach of anyone, particularly a stranger, to the bed causes a scream of fear. Finally, the pain is sufficient without touch to occasion whimpering or crying even in sleep, in so far as this is retained. In the course of a few days a deep, tense swelling appears upon the thigh, usually at the lower end or passing from there to the shaft, and gives to it a spindle shape. As a rule the swelling is not

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<sup>1</sup>The great group of American statistics, to which we shall frequently allude, cannot be utilized here. Reports of this collection will be found in *The British Med. Jour.*, 1898, II, and *Jahrb. f. Kinderheilk.*, Bd. LI.

sharply defined and is immovable. The overlying skin may be adherent and present a tense and glistening appearance. Redness and heat are absent. The circumference of the thigh may be increased decidedly.

In rare instances there are evidences of infraction. In contrast to disease of the thigh, which is always present in typical cases, painful swellings of the lower leg, especially at the upper end of the tibia are rare, and usually appear simultaneously with the hip affection. In the arms tenderness and pseudo-paralysis are uncommon; swelling also is often absent notwithstanding intense pain. The local and functional disturbances are slight. Less frequently there is implication of the ribs, the vertebræ, and the bones of the skull. Under two circumstances a very peculiar picture arises: When all of the true ribs near the boundary of the cartilaginous bone are bent the sternum sinks as if it were pressed inward; when the walls of the orbit are involved the eye-ball is protruded. This protrusion soon affects both eyes, without marked limitation in movement, and may at intervals increase.

In contrast to 47 cases of disease of the thigh, Heubner saw the lower leg attacked 21 times, the bones of the orbital cavity 4 times, but rarely the forearm and ribs. I found the thigh attacked 35 times, the lower leg 7 times, the plate of the orbital cavity 4 times, the arms 10 times—in most instances reported by the parents—and the ribs once.

The swelling, even when the skin is involved, does not produce a true edema; on the contrary, when there is swelling of the lower leg the edema appears at the end of the tibia or on the foot. The swelling over the bones indicates hemorrhage under the periosteum; the hemorrhage may, however, also involve the subcutaneous tissue. This is noted most frequently in the eye, where, prior to or simultaneously with the projection of the eye-ball, a swelling of the upper or, more rarely, of the lower eye-lid usually appears which is caused by hemorrhage, as is shown by the later discoloration. Hemorrhage—punctiform or larger—into the skin alone, without simultaneous deep hemorrhage, is rare (7 times in 40 of my cases, 7 times among Heubner's) and hemorrhage into the mucous membranes is also uncommon; I once saw hemorrhage under the conjunctiva. On the other hand, the mucous membrane around the *teeth*, or at their points of insertion is usually affected soon after the onset of the disease so that a normal mucous membrane in this region is unusual (in 50 of Heubner's cases the mucous membrane around the teeth was found normal 6 times, and among 23 cases I found a normal condition 5 times). At first there is slight redness; later this changes to a bluish tint and the mucosa becomes constantly more tumescent until it covers the tooth like a blue vesicle. This vesicle may rupture at its summit and give rise to more or less severe hemorrhage, while at the point of rupture an ulcer is formed which, according to Barlow, may undergo decomposition. This disease of the mucous membrane of the teeth is usually more marked upon the upper jaw.

Clinically, the internal organs are less involved. *Disease of the kidney* is of greatest importance. In very rare cases—in a severe general affection—

there is an irritation of the kidney with a moderate amount of albumin and a few casts. Hemorrhage from the kidney is more frequent; the urine may be tinged with red, but commonly it shows a fresh red discoloration. It contains numerous red blood-corpuscles, occasionally cylindrical, scant hyaline or granular casts, which may be covered with red blood-corpuscles and renal epithelium, moderate amounts of albumin (the highest quantity I found was 1.4 per 1,000), and a sediment of uric acid salts. Heubner found "hemorrhagic nephritis" 6 times in 65 cases. In my cases, according to my own observation or the analysis of the urine, blood was absent 12 times (in another series of cases nothing can be stated in regard to this, and once the nephritic urine was free of blood), while in 7 cases I was able to demonstrate hemorrhage.<sup>1</sup>

Hemorrhage of the intestinal mucous membrane is rare and shows itself by a trace of recent blood or hemorrhagic mucus in the feces with occasional irritation of the colon. Hematemesis is uncommon.

Enlargement of the spleen is so extraordinarily infrequent that its connection with Barlow's disease is not positive.

Convulsions due to the pressure of subdural hemorrhages (Cassel) have occasionally been reported.

Simultaneously with the development of the disease in the bones and in the teeth there is impairment of the general condition, usually preceded by constipation or hard feces. Ingestion of food becomes difficult, if it has not already suffered prior to the outbreak of the disease. At first the child is eager for food but after a few attempts to suck, the nipple is forced away by the tongue, *the child turns its head aside and closes its mouth firmly*, or beats the bottle with its hands. Even the sight of the bottle induces crying and not until the child is sleepy is it at all possible to give nourishment with a spoon. Naturally this impairs nutrition.

Sweating of the entire body, or at least of the head, is an almost invariable occurrence. Increasing debility, and in particular a general pallor of the skin, which may become extreme, are noted, as well as very irregular fever, the exacerbations of which are in association with renewed hemorrhage. Among 24 cases, the temperature of which was taken by myself, fever was present in 15. Cassel noted a febrile temperature 7 times in 11 carefully investigated cases.

However, it must be expressly emphasized that the development of the clinical picture follows no definite law. There are cases in which the pallor, notwithstanding well-defined symptoms, is so slight that its recognition must depend upon the mother, who can make a comparison with the former condition. Inversely, I noted a decided sallowness of the face and severe cachexia in a child whose ribs and legs were very sensitive but who showed no distinct swelling nor the slightest change in the gums (fracture of the thigh occurred during examination by a physician).

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<sup>1</sup> See conclusion of article.



As a rule, in the typical cases the first examination of the child reveals pain and swelling in the legs, and, in so far as teeth exist, disease of the gum. The development of other symptoms is variable; sometimes the bones are most affected, at other times the constitutional condition; in rare cases there is a decided tendency to cutaneous hemorrhage. If the deleterious factors which are the cause of the disease continue uninterruptedly a fatal outcome may be expected. Exceptionally the lethal termination has occurred in three or four weeks after the appearance of the typical symptoms (Naegeli); death seldom occurs before the seventh or eighth week and usually the disease lasts three to four months. The individual case may show an irregular severity. These fluctuations, and with them the protracted course, are probably due to a temporary diminution or abandonment of the deleterious agent. For the same reason there is no definite law in regard to mortality. Recovery—in the absence of complications—or death is dependent upon the decrease or continuance of improper nourishment. That 15 to 20 of every 100 of these cases have perished must be ascribed to an insufficient knowledge of the cause of the malady.

The foregoing facts relative to a purposeful or accidental removal of the cause also indicate that the symptom-complex attains a varied development. In the prodromal stage—with pallor or rejection of food—the outbreak of infantile scurvy may be prevented, or after the first indication of a symptom the process can be aborted, by a change of nourishment. For example, in a flourishing infant I was able to arrest the disease before symptoms other than sensitiveness of the legs (without swelling) had appeared. Hematuria may exist for some time as an unassociated symptom, without sensitiveness or swelling of the gums. Among 45 cases I saw 5 abortive types of this kind. On the other hand I have only twice found hemorrhagic urine in well-defined clinical pictures of the disease.

If the malady is brought to a favorable termination by a proper change in the nourishment, the pains vanish with surprising rapidity—in my experience as early as one or two days—the inclination for food increases constantly, and the quiet child now takes nourishment well and appears greatly improved. The gingival swelling recedes without local treatment and in eight or ten days the gums may be normal. The cutaneous hemorrhages run their color scale and disappear. The swelling of the bone subsides somewhat more slowly and may require some weeks for its complete extinction. The hematuria usually ceases in a few days but after a protracted course it may continue for weeks. As a rule, not many weeks after the institution of treatment the child may be regarded as cured.

## PATHOLOGY

The gross *anatomical foundation* of the clinical picture has long been known. Its refinement has to some extent been accomplished in the last few years by extensive investigations (Naegeli, Schoedel and Nauwerck, Schmorl, v. Recklinghausen, Jacobsthal, Ziegler). The swelling on the bone is due

to copious effusions of blood which commonly cause a decided upheaval of the periosteum and undergo involution in the usual manner. If new hemorrhages occur with the old, the organized blood-clot produces a stratified structure.

The osseous hemorrhages which occur in the bone-marrow as well as under the periosteum, together with hemorrhage into the muscles, into the subcutaneous tissue, and into the internal organs, are the manifestation of a *hemorrhagic diathesis*. No sufficient explanation for this is found in the blood. Its morphologic examination in life has shown only slight change in the red blood-corpuscles corresponding to a moderate anemia, and the leukocytes are certainly not increased, although perhaps there is a relative preponderance of lymphocytes. On the other hand, peculiar to infantile scurvy are the changes in the *bones*, which are distinct from the possible rachitic changes. In the diaphysis of the bone in particular the normal bone-marrow has been transformed into a gelatinous and fibrous material, the osteoblasts are scant, the normal ossification arrested. According to Ziegler there is also resorption of bone. The bony trabeculæ are therefore thin and scant. For this reason fractures readily occur in the new and thinnest trabeculæ—hence below and not within the cartilagino-osseous border. In advanced atrophy of the bone, however, fracture is more frequent toward the middle of the diaphysis, and the decided displacement of tissue which then results gives rise to hemorrhages, or augments those already present.

Fractures and hemorrhages are produced by the slightest mechanical influences. The first and most marked appearance of the affection in the legs is readily explained by their greater use at the time of life in which the disease occurs (Michael Cohn): It is due to the stamping and striking of the legs, attempts to sit, stand, and walk, as well as to slight torsion and shock (drying the child, bathing, stumbles, falls, etc.). Cutaneous hemorrhages are produced by slight pressure or blows. Hemorrhage into the eye-lids and orbital cavity have sometimes been the result of prolonged crying. The affection of the gums is due to the pressure exerted upon the mucous membrane by the teeth in dentition and perhaps also to external causes, as biting. The use of the already erupted teeth produces an irritation which, as upon other occasions is usually greater upon the upper jaw. It is a notable fact that in the attack on the osseous system the joints are not implicated. Inflammatory symptoms do not appear.

Anatomical investigation provides no explanation as to the cause of the disease. The hemorrhages are not characteristic, although their preference for the bones and gums resembles scurvy. The peculiar affection of the bony tissue cannot be utilized in this connection because corresponding investigations in epidemic and endemic scurvy do not exist. Nor does the clinical picture give enlightenment, particularly as endemic scurvy has not been observed in nurslings. Although pathologists do not yet coincide as to a connection of infantile scurvy with rickets the clinician may at once reject such an assumption. Scurvy is seldom observed in rachitis, and rachitis as a rule,

does not accompany infantile scurvy. On the other hand, sufficient explanation is given by etiologic research; the correctness of the result is proven by the success of treatment, which is founded upon it.

## ETIOLOGY

Infantile scurvy is observed only in children who have been artificially fed for several months. Neither Barlow nor any other competent recent investigator has reported a contrary experience, and although under quite exceptional circumstances the disease is noted occasionally in a breast-fed child, the majority of the few such reported cases are certainly mistaken.<sup>1</sup> Although the harmful agent undoubtedly lies in the quality of the artificial food, nevertheless we must be cautious in our decision. The monotony of the food is occasionally mentioned as the cause, but this view may be rejected, since there is no greater variation when natural food is administered at the same period of life. Simple under-nutrition also does not produce infantile scurvy.

The food which is at fault consists mostly of milk in certain preparations, infant foods, or a combination of both. In some cases other food has been added, the influence of which will be explained later. Many communications mention only the food product, in the belief that the deleterious element is thereby designated, and say nothing of its mixture with milk or the manner in which the milk is prepared. In other instances the administration of milk is mentioned, but through ignorance, or because of certain theories, the nature of its preparation is not reported. Finally, the author has not always known that any prolonged method of feeding was without importance, but that the food which was chosen for a number of months prior to the disease is of moment, whether administered uninterruptedly, or alternately with another which was just as objectionable. For all of these reasons many reports in the literature are valueless in the etiology, and many of the objections to the theory which is to be developed are untenable. Therefore, in this discussion I must depend largely upon my personal experience.

First, we may assume that exclusive feeding with prepared infant foods which do not contain milk—apart from some unimportant mixture, as tea or broth—may develop infantile scurvy. I am not aware of any positive case of this kind in German literature which has been fully detailed; but in England and America several such observations have been reported (for example, Case

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<sup>1</sup> In some of the cases which have been much quoted a period of artificial nutrition covering several months intervened between the period of natural feeding and the outbreak of the disease. I saw a breast-fed child, aged 5 months, who, after the first month, was occasionally fed condensed milk. This child developed tenderness of the legs and hemorrhagic nephritis. In the following week there was a varied amount of blood in the urine, with scant granular and hyaline casts, and a quantity of albumin equal to 4 per 1,000, later 2 per 1,000. Although this was a case of nephritis, there was an undeniable resemblance to infantile scurvy. This sprightly child, who continued to be nourished at the breast, had traces of albumin in its urine for over two months.



I of Cheadle,<sup>1</sup> *Lancet*, 1878, Nov. 16. In those countries, therefore, the affection is not rare, after the prolonged use of infant foods—whether or not they contain milk in addition to carbohydrates. Milk products in condensed or liquid form which are partially prepared by energetic chemical manipulation, partially by high heat, are a further cause. Finally, infantile scurvy is observed after administration of uncombined milk, which, with or without the addition of mucilage, flour, or sugar, has been heated for a certain time in manufacture or at home. In Germany examples of this kind are numerous. In this condition a great rôle is played by “sterilized” milk—a designation which fails to define the duration and degree of heating: On the one hand we must consider milk which is decomposed in its manufacture, as is shown by the brown discoloration, on the other hand milk which has only been boiled in the Soxhlet apparatus for a certain time. Just as little is conveyed by the expression “Pasteurized milk.” Milk of this kind has been heated at various high temperatures for an indefinite period without being brought to the boiling point. In the year 1901, Pasteurized milk, which is mentioned in the following observations, was heated to a high temperature (95° C.-203° F.); since 1902 it has been heated for half an hour at a temperature of 60° to 65° C. (140° to 149° F.). The following reliable investigations are examples of the degree of heat to which the milk is sometimes subjected. In Cassel's experience the milk was in two instances sterilized excessively, twice Pasteurized milk was heated in the house from 20 to 30 minutes, and nine times fresh milk was heated in the house from 20 to 60 minutes. I have observed the following methods of heating: Twice, *sterilization* in manufacturing and at least once boiling at home for three-quarters of an hour; Pasteurized milk heated additionally in the Soxhlet apparatus, once for 45 minutes, three times for 20 to 25 minutes, in a milk cooker, twice for 20 minutes, in the Soxhlet apparatus, three times for 15 minutes, once for 10 to 15 minutes, twelve times for 10 minutes, in milk cooker, once for 10 minutes; in one case, 15 minutes in the Soxhlet apparatus for 3 months, and during the following 3 months 7 minutes; in one case 10 minutes for 3 months and 2 to 3 minutes the following 5 months; in two cases 4 minutes and 1 minute, respectively (by the watch). Condensed milk was given in one case for 3 months, and in the following 5 months Pasteurized milk was boiled in a casserole for a few moments. In another case plain milk for 8 months, then Pasteurized milk boiled in the Soxhlet apparatus 12 minutes for 2 months. In another case fresh milk boiled 15 minutes for 2 months and 10 minutes for 3 months, then for 6 months Pasteurized milk boiled in a Soxhlet apparatus 10 minutes. In three cases fresh milk was boiled in the Soxhlet apparatus 15 minutes, in two cases 10 minutes. The following case is noteworthy: For 3 months Pasteurized milk was boiled in the Soxhlet apparatus 5 to 6 minutes, then, for 6 months, fresh milk was boiled for half an hour at a temperature of 70° C. (158° F.)

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<sup>1</sup> Here, in addition to oatmeal, “rusks,” i.e., zwieback, which may have been prepared with milk.

and kept at that temperature with the thermometer. It is to be remarked that in neither of the above cases in which Pasteurized milk was cooked in the Soxhlet apparatus for 4 minutes and 1 minute, respectively, could a boiling temperature have been reached<sup>1</sup> so that not boiled milk but milk heated under 100° C. (212° F.) may have a deleterious effect, as is shown by the American statistics.

Improper nourishment must be prolonged for months before infantile scurvy arises. According to my observations, children who have had improper food from their birth have shown unmistakable symptoms most frequently between the seventh and eighth months (17 times), more rarely between the fifth and sixth months (11 times) and exceptionally between the ninth and eleventh months (8 times). If infantile scurvy develops later the damage was not begun at birth but a greater or less time afterward and continued for at least five months. For example, I saw a child affected at 13 months, which was fed at the breast for the first 5 months; another child was attacked at 20 months who, instead of having raw milk was for a few months given Pasteurized milk from the same dairy, which was boiled in the Soxhlet apparatus. Literature furnishes numerous examples of this peculiar regularity in the outbreak of the disease. One of the oldest children affected with infantile scurvy was a child aged 2 years and 9 months, who had been nursed until 2 years of age, and had then been fed bread, butter, tea, sausage occasionally, and to some extent brandy and water (Cheadle).<sup>2</sup> In general this affection is not found much beyond the second year because after that age a mixed nourishment is given.

The kind of food which produces infantile scurvy has been mentioned, and, to prevent misunderstanding, I must reiterate that only when this form of nourishment has been continued for an average eight months does the disease develop. The greater the decomposition of the food from chemical effects or from heat, the more rapid and severe in general is the onset. This is especially evident in the use of artificial or intensely heated milk preparations, such as albumose and fat milk, which give rise to such severe grades of anemia as rarely exist when the milk is prepared at home.

On the other hand it seems peculiar that Barlow's disease has arisen—exceptionally, it is true—from the use of prepared milk under which many other children have remained perfectly well. The antecedent history of these cases occasionally reveals a previous mild or severe intestinal disturbance (dyspepsia, intestinal catarrh).<sup>3</sup> In other cases only severe constipation could be discovered, which is very usual after the ingestion of milk that has been

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<sup>1</sup> According to the investigations of my assistant, Dr. Forest, when milk in the Soxhlet apparatus is at the same temperature as the water, it attains the temperature of boiling water five minutes after the water has begun to boil; if the bottles are in water only 1 cm. deep nine minutes are required.

<sup>2</sup> *Lancet*, 1878, Nov. 16.

<sup>3</sup> In the history of 39 cases I found dyspepsia and intestinal catarrh 12 times, and in 5 instances other important affections.

cooked for some time. Apart from these factors, however, there is apparently an obscure *individual predisposition* which must be invoked, particularly in those cases in which the nutritive damage is slight. Finkelstein reports the case of a child who, as its brother had previously been attacked with infantile scurvy, was fed with boiled milk and vegetables, but nevertheless contracted the malady.

In the main, however, we must look for the cause of Barlow's disease in exclusive and long-continued nutrition with certain foods which have undergone a decided change, particularly from overheating. This also satisfactorily explains the temporary and local dissemination of the disease. The avoidance of natural nourishment in the last decades is not responsible for its increase, for infantile scurvy was also unknown in districts in which breast-feeding was not unusual, and where even now the attacks in artificially fed children are infrequent. On the contrary, the appearance of infantile scurvy has to do with the theoretic and practical evolution which the problem of artificial infant feeding has of late undergone. In the last decades, with a better knowledge of the chemical composition of milk, the production of nutritive preparations as substitutes for milk has enormously increased. Furthermore, the dangerous intestinal catarrhs, which had been recognized as due to the decomposition of milk, since about 1880 have been known to originate from bacterial contamination. An attempt was made to overcome this contamination by prolonged boiling. Although this procedure has been somewhat restricted in the last few years, there has been a development in another direction, in that the milk is now subjected to Pasteurization at the dairy. This alone, or after additional heating at home, may have a deleterious effect.

These facts are in conformity with the more frequent occurrence of infantile scurvy, especially in England and in the United States, where infant feeding in the direction above indicated has been particularly developed. From 1889 to 1894, 106 cases were reported, and the collective investigation undertaken in 1898 gave 379 cases which were referred essentially to this form of nourishment. The disease is also common in Holland, especially in Friesland—de Bruin collected 61 cases in 1893—but information as to the nutrition of the diseased children is wanting. There are few cases in France and in Switzerland, notwithstanding the frequent employment of sterilized milk in those countries, but it remains questionable whether milk of this kind is often fed exclusively for five months, and upon the other hand, whether the knowledge of infantile scurvy is sufficient to establish a diagnosis. In Germany, Rehn, who was the first to devote special attention to the disease in that country, saw only 7 cases from the year 1878 to 1889. Following Rehn, Heubner interested himself particularly in the affection but from 1876 to 1889 saw only 2 cases; from that period up to 1900, 28 cases. As the result of special inquiry in Schleswig-Holstein v. Starck reported a number of cases of infantile scurvy which were due to prolonged boiling of the milk in the Soxhlet apparatus; more important, however, was his discovery in Hamburg of a small epidemic which was proved to have arisen from the feeding of milk sterilized in



the laboratory. In Berlin, up to the year 1900 inclusive, infantile scurvy was comparatively rare, although the manufacture of albumose milk brought forth on occasional augmentation. Meanwhile, from the year 1896 to 1900 Heubner, Cassel and myself together saw only 32 cases, while from that period the number increased suddenly to such an extent that in the years 1901-2 the same observers personally treated 83 cases. According to my own experience this increase is to be referred to the ingestion of milk which was Pasteurized in the dairy and afterwards heated at home, and its suddenness is explained by the fact that the milk of the dairy in question was not Pasteurized until 1901.<sup>1</sup>

The prolonged use of infant foods and prepared infant milk is expensive; the possession of a Soxhlet apparatus requires an outlay too great for the poor. This is the reason, set forth by all authors, that infantile scurvy occurs only among the better population. In Berlin the children of the well-to-do middle class in particular are affected. Here, owing to professional or popular advice, we see the over-careful mother—possibly with a watch in her hand—accurately follow the prescribed time for boiling, or even prolonging it from a theory of her own. In support of older professional views she is careful not to allow her child other food than the milk until a definite period. Exceptionally, the disease is found also among the poorer class, who attempt beyond their circumstances to provide the best for their children. As a rule these children are all well-nourished and free from hereditary rickets. On the other hand, a small number of poor children who are sickly and rachitic are affected under special circumstances with infantile scurvy: in a hospital for a long time, and fed according to the newest artificial methods, the reverse effect of these measures shows itself in the form of infantile scurvy; or they are nourished continuously and exclusively with food preparations or over-heated milk for some accidental reason, especially to prevent a renewed intestinal catarrh. These children supply the material for the latest anatomical investigations; more or less rachitic, they have been the cause of the unending dispute as to the connection of infantile scurvy and rickets.

It was surprising that the affection here described was at first declared to be *scurvy*. Scurvy ordinarily appears in epidemics, among the crews of ships, or among a starving population; young children are not mentioned among the patients;<sup>2</sup> probably those in the first year of life—provided they are not breast-fed—succumb from the unhygienic and economic conditions which predispose to scurvy before the disease can develop. That small children may develop typical scurvy is shown by the cases from the older literature of the disease under consideration.<sup>3</sup> More essential is the fact that in the endemic scurvy of adults, as well as in the typical cases of childhood, the symptoms do not deviate

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<sup>1</sup> Among 26 cases treated during this period by me 23 had been fed with this milk for some months. The previously mentioned cases in the year 1903 also included numerous examples.

<sup>2</sup> See *Inaugural Dissertation of Tschudakoff* (Berlin, 1901), who reported 1,153 cases of scurvy in Russia in the years 1898-99.

<sup>3</sup> Cases 1 and 3 (the latter quoted above) by Cheadle, *Lancet*, Nov. 16, 1878.

fundamentally from the affection which has been described: in every case there is anemia, swelling and painfulness of the legs, with more or less flexion and outward rotation,<sup>1</sup> swelling and ulceration and even putrefaction of the gums. Apart from the influence of certain physiologic conditions, as were shown above, upon the localization in nurslings, *infantile scurvy appears peculiar only in so far as it shows the symptoms of scurvy alone*—without simultaneous emaciation or other disease due to improper nursing and nutrition—and *moreover is not developed to the extreme extent*. On the contrary, the children are carefully looked after, plentifully nourished and as a rule well developed. Hence infantile scorbutus, as a *pure* form of scurvy, is especially suitable for study of the nutritive conditions, and therewith must be considered that form of nourishment by which curative results are positively obtained.

As in endemic scurvy, so also in the infantile form, the predisposition is individual. It cannot be denied that with a marked predisposition even a slight deterioration of food (sometimes from the breast) may produce cachexia with a tendency to hemorrhage; *but it will probably at no time be a case of simple inanition*. In infantile scurvy the good nutrition, which is the rule, shows that there is neither a decreased ingestion of food nor a special limitation of absorption and assimilation. If super-heated milk is to be regarded as a cause of scurvy, a careful weighing of all investigations shows its digestibility to be little or not at all limited. Even though organic combinations of phosphorus, the presence of which is necessary for the accumulation of tissue<sup>2</sup> appear to be decomposed by heating, this loss is not of importance, particularly as prolonged additional feeding of an uncoagulated egg—nothwithstanding a plentiful presence of lecithin—is not capable of preventing the development of infantile scurvy. We may therefore disregard a possible damage of nutrition and investigate as a causative factor the general constitutional disturbance which is present in infantile scurvy.

The fundamental and also practically important question is whether scurvy is due to the *absence* of products which, although not necessary for a gain in weight, nevertheless are essential for development, or whether it arises from the *presence* of substances which are injurious. To confine our remarks to milk, modern physico-chemical methods of investigation, in spite of their minuteness, show neither a decrease of the molecules (perhaps through separation upon heating) nor an increase of the same which might follow the splitting of salts from their union with organic combinations.<sup>3</sup> Therefore there can be only minute changes in the milk through heating. If we consider the *disappearance* of products by cooking all action of ferments will unquestionably be arrested at the boiling temperature—so rapidly, indeed, that even in milk

<sup>1</sup> Compare a characteristic picture of a woman in "Starving Russia," by C. Lehmann and Parvus. Stuttgart, 1900, p. 309.

<sup>2</sup> Cronheim and Erich Müller, *Zeitschr. f. diätet. und phys. Therapie.*, 1902-3, Bd. VI, Heft 1-2.

<sup>3</sup> Compare H. Neumann, "Remarks upon Barlow's Disease." *Deutsche med. Wochenschr.*, 1902, Nr. 35 and 36.

which is boiled by the ordinary method they will never be present: Their absence, therefore, cannot be of importance. Less calcium will be absorbed in the intestine from boiled milk than from raw milk; and apparently the metabolism of the calcium<sup>1</sup> also suffers: But that this has to do with infantile scurvy, and not perhaps with rickets, must first be proven. We might more readily observe that the introduction of iron from cow's milk, as to amount or in a tissue-producing form, is damaged by heating: According to Bunge the danger of a decrease in iron and of anemia exists even with natural food, so that if newborn animals are fed too long upon milk exclusively such a fear is certainly justified when the very slight quantity of iron in the cow's milk is still further decreased by heating. It is true, boiling does not precipitate noteworthy amounts of iron from cow's milk and it must also be remarked that with the use of boiled milk exclusively neither the addition of organic nor of inorganic iron combinations can prevent or cure scurvy. Furthermore, infantile scurvy may develop with a continued and exclusive ingestion of meat, notwithstanding the fact that there is then no deficiency in iron.<sup>2</sup> Finally, Netter's view must be mentioned,<sup>3</sup> that the citric acid present in the milk is precipitated by boiling and forms a combination difficult to dissolve. Granted that cow's milk contains about twice as much citric acid as human milk, boiling may all the more readily lead to a perceptible deficit, as the quantity of citric acid varies according to the individual, season, etc. While Netter regards citric acid as the antiscorbutic portion of the milk, he refers to the specific action of the vegetable acids contained in vegetables. This hypothesis, however, appears erroneous, for the reason that, in the Arctic region for example, man may remain perfectly well with an exclusive diet of fresh meat, without milk or vegetables. On the contrary, we are inclined to the view that in the case of scurvy toxic products are mediately or immediately taken up in the economy which—perhaps through internal secretion—are not rendered inactive in the juices of the body, or at least to a sufficient extent. The anti-toxic faculty of the tissues and their juices may vary individually, and besides may be decreased by preceding disease, especially of the intestine. With a prolonged ingestion of toxic products such as are contained in spoiled preserved meats or in meat that is not fresh, there may be a development of scurvy—as we know from its occurrence at sea. Johannessen's view that toxins which originate from dead bacteria in the milk may act in the same manner, cannot be admitted in the case of infantile scurvy, for as a rule this affection arises after the ingestion of milk which is free of bacteria. On the contrary, when we consider the development of infantile scurvy after the use of flour and milk preparations and of too greatly heated milk, we must remember that the albumins in the flour and in the milk have been exposed to chemical action or to the effect of heat, and have thus undergone more or less decomposition or at least a change in their intramolecular combination. It has been

<sup>1</sup> Cronheim and Erich Müller, *l. c.*

<sup>2</sup> William P. Northrup, *l. c.*, Case 8.

<sup>3</sup> *Bull. d. l. Soc. de Pédiatrie de Paris*, Octobre, 1902.



proven that in too greatly heated milk, apart from the destruction of other constituents, split products of albumin (leucin, tyrosin, sulphuretted hydrogen) are present, and also that under artificial digestion deleterious products, such as resorbable ammonia, may be formed in large amounts (G. Klemperer). *Therefore scurvy may, with some likelihood, be regarded as a more or less chronic intoxication,*<sup>1</sup> whether the poisons develop in an exogenous manner in the food, from bacterial decomposition, through chemical action, or from the effect of heat, or whether they originate endogenously in digestion. Besides, *according to the nature of the food and the degree of nutritive damage, various deleterious products may become prominent and influence the clinical picture;* at least it is conspicuous that the marked change in albumose-milk, and in greatly super-heated fat-milk because of transportation, may produce severe anemia and cachexia (in one of my cases hemorrhage occurred without a manifest cause), while in many other instances nourishment with milk heated in the Soxhlet apparatus produces the characteristic hemorrhages but not marked anemia and cachexia. We have already found that there is apparently no immediate anatomical connection between the changes of the bones and their marrow and the hemorrhagic diathesis.

## DIAGNOSIS

Having gained an insight of the picture and nature of infantile scurvy we are better able to consider the *diagnosis*. As the knowledge of this disease is not extensive, a correct diagnosis is rarely made. The confusion which arises is of a surprisingly manifold nature. Very frequently a single symptom only is observed; often also many symptoms are combined and thereupon an incorrect diagnosis is made. The disease of the gums is usually referred to difficult teething, and is therefore considered of no great importance. We are accustomed to look upon this as an unavoidable annoyance, and hence, in a moderate affection, it is thought that there is no connection with the pain in the legs. If, in considering the hemorrhages, the swelling is regarded as the essential and the sensitiveness is overlooked, tumor formation is thought of; in the thigh osteosarcoma, in the eye a retrobulbar tumor; more than once the swelling, the nature of which was not clear, has been attacked surgically. If the painfulness in the bones is heeded, particularly when fever is present, it is natural to think of inflammation—periostitis or osteomyelitis; in favor of the latter also is the multiple localization and the course, which is sometimes protracted, sometimes rapid. The presence of albumin in the urine would thus be explained. It is true that at the onset of the swelling there is rarely fever,

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<sup>1</sup> The very rare cases of infantile scurvy which occur in breast-fed children—in so far as they are not at all positive—must be tested from this point of view. I know of only one case, that of Love (*Journ. Amer. Med. Assn.*, October 12, 1895). Here the mother, who also was debilitated, and the child, aged 9 months, were subjected to strong mercurialization, so that the case may have been one of severe mercurial intoxication.

which is so apt to be present in an inflammatory process, and, on the other hand, if the swelling is of long standing its stationary condition is opposed to inflammation. A certain similarity with the painful swelling of the bone in infantile scurvy is found in syphilitic osteochondritis when associated with periostitis and infiltration of the soft parts (Parrot's pseudo-paralysis), but it does not pass to the shaft, affects principally the upper extremities, and above all it appears at the earliest stage—only exceptionally between the seventh and ninth months. Simultaneously, as a rule, there are other signs of syphilis. The spontaneous fractures of infantile scurvy may resemble simple traumatic fractures, but the external causes—if such can be demonstrated, are clearly of a disproportionately mild nature. Often the sensitiveness and swelling impress the physician less than the limitation in movement caused by the pain; or there is an undefined conception of a nervous or of a trophoneurotic disease, and a diagnosis is made of hysteria (!), of cerebral hemiplegia, of infantile paralysis, of myelitis. It is unnecessary, however, to call attention to the differential diagnosis from the partial pseudo-paralysis of infantile scurvy. Further, in a consideration of the pain, neuritis, coxalgia, and a masked disease of the vertebral column are suggested. Perhaps the most common confusion is with rheumatism, but exact investigation will prove that in infantile scurvy the joints, although at times transitorily fixed, are always uninvolved. Exceptionally—in children with severe rachitis—the sensitiveness of infantile scurvy may be confused with the bone pain of rickets, but in severe, florid rickets the thorax is very sensitive, and at the same time the long bones are more or less flexible—a condition which is rare in infantile scurvy. Also, in the latter affection the swelling of the bones is not marked except at the epiphysis, provided there are no infractions with formation of callus. Finally, in so far as the age is not against them, there may be confusion with erythema nodosum and with septic affections and diseases of the blood (especially leukemia), in which there is a tendency to hemorrhage. The diagnosis of renal hemorrhage is especially difficult when it occurs as an isolated or principal symptom of infantile scurvy. If there are simultaneous periosteal hemorrhages in the legs, the edema which appears below, upon the lower leg and the foot, should not be confused with nephritic dropsy. If hematuria exists alone, the ordinary bright red, instead of brownish, tinge of the urine, the variation in the quantity of blood, the few casts and the limited amount of albumin should at least awaken a doubt of a renal affection, especially as at this age a causal factor of renal inflammation is usually absent.

It cannot be denied that at the onset and in the abortive types of infantile scurvy, as well as with the simultaneous existence of another disease, the diagnosis may be very obscure. From the moment, however, at which the possibility of this rare affection is presented the picture clears rapidly, for if a careful history is taken, especially of the nourishment during the last five months, we will know at once whether infantile scurvy need be brought at all within the scope of differential consideration. While the examination must be very complete, nevertheless, in the interest of the child a certain restraint must

be imposed: Fracture of bones has several times occurred during examination, also orbital hemorrhages from crying, due to the pain, which may suddenly protrude the eye-ball. Occasionally, after every examination the sleep of the succeeding night is disturbed. The sensitiveness of the leg generally indicates the diagnosis, and after a careful inspection of those members, and the external examination of the remainder of the body, the condition of the internal organs must be determined, though usually they reveal nothing of importance: the pharynx, mouth, and particularly the gums must be inspected and the temperature taken in the rectum. It is often difficult to obtain the urine for purposes of examination because of the pain when the child is held. If convulsions are present—a rare occurrence—external bleeding of the skull will be found, which indicates intracranial hemorrhage. Fortunately, if a positive diagnosis is impossible, the treatment—a complete change of diet—will enable us within a very few days to come to a final conclusion.

### TREATMENT

There is scarcely another affection in which the *treatment* is so rapidly and certainly effectual. In infantile scurvy, in contrast to the epidemic disease, the external conditions are always so favorable that the necessary treatment can easily be carried out. *The first point of importance is to stop the improper nutrition:* Milk and infant foods might be altogether omitted, but in those cases where only the too prolonged boiling of simple cow's milk has been operative the degree of heating may be changed. Under some circumstances a certain limitation is sufficient: A shorter period of heating in the Soxhlet apparatus, boiling up two or three times over an open fire when Pasteurized as well as cooked in the Soxhlet apparatus, or Pasteurization alone. This change in the heating of the milk is sometimes sufficient to produce a cure; the physician must know, however, before giving his orders, whether, as is usual nowadays, the milk to be used has been previously warmed in any way in the dairy. To a lack of this knowledge was due the only fatal case in my practice. We have observed that moderate warming is not always well borne by weak children; under such circumstances it is better to bring about a change by the avoidance of heat. As a matter of fact the most immediate results are obtained by the employment of raw milk. It may be given undiluted in amounts of 800 to 1,000 grms. daily, slightly warmed. The milk must, however, be obtained from healthy cows and must be free of the germs which cause infectious diseases in the human being, or that give rise to saprophytic decomposition. Raw cow's milk is commonly taken with great relish—in contrast to the milk previously administered—but we must be careful to observe that it is well borne and does not cause or increase diarrhea. In addition to the products which are especially conducive to health the milk may contain those which are foreign to the human body and which would be destroyed by boiling. Thus, after the administration of raw milk I saw a severe erythema exudativum multiforme appear.



If the milk in its raw state is not borne even when diluted, it should be warmed for a short time over an open fire. The addition of the ordinary infant foods does not hinder the curative process, which is a proof that the incorrect method of heating the milk is the cause of infantile scurvy rather than the addition of infant foods. Whether the beneficial result of raw milk is due to the absence of the deleterious products of heated milk, or to the introduction with the raw milk of antiscorbutic substances, such as citric acid, or perhaps to both of these causes, cannot be stated definitely. In favor of the first view is the partial or non-success which attends the use of antiscorbutics with simultaneous improper milk feeding.<sup>1</sup>

From the emphasis placed upon the curative effect which is brought about by the withdrawal of the deleterious food, it is evident that the administration of remedies which are specifically antiscorbutic are not necessary in infantile scurvy. Nevertheless they may be useful. When there is a constitutional change in the blood which is manifested by a tendency to hemorrhage these expedients are calculated to bring about an improvement in the tissues. Even in a case of congenital hemophilia I saw success follow treatment with lemon juice. Just as little as the damage wrought by scurvy to the physical condition, and especially to the hematopoietic organs and the blood, can be referred to a uniform chemical agent, do the remedies employed for scurvy uniformly remove the chemical cause of the affection. The familiar antiscorbutics are of various kinds. On the one hand, in addition to milk and honey, there is *meat*; its salts are absolutely without effect; *meat-broth can never prevent the development of infantile scurvy nor cure it*. Probably, therefore, genuine albumin is the active principle; the expressed juice of fresh meat is administered, several teaspoonfuls throughout the day, and observation is made as to whether or not it is well borne. In contrast to this, *eggs* are absolutely without effect, although they contain abundant genuine albumin and other substances which are essential for the growth of the body.

Of the *vegetables*, those containing seed are ineffectual, in whatever form they are prepared. It is an error, however, to regard green vegetables alone as specific antiscorbutic remedies, and to believe that they must be fresh when eaten. The active vegetable substances require heat and are found in vegetables of the most varied kinds. That we are here dealing in part with *vegetable acids* is shown by the use of lemon and orange juice. Other fruits will act in a similar manner, whether raw or cooked, for example, apples, pears, gooseberries, whortleberries, strawberries, and the like, peaches, apricots, plums, cherries, pineapples, melons and dates. Among the vegetables may be mentioned spinach, carrots, peas, kale. The action of asparagus and cauliflower is doubtful. The various salads also come into question—cress, lettuce, green salad—and stewed rhubarb. Mashed potato is recognized as a valuable antiscorbutic food.

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<sup>1</sup>Case of Finkelstein and other reports in literature. As to the possibility of a further view see later!

*The action of vegetables is noted only when they are taken in sufficient quantity* (several teaspoonfuls of the juices, especially lemon and orange juice, and at least a small dish of the vegetables); the administration of a few teaspoonfuls is not enough to prevent infantile scurvy.

In place of the food hitherto administered it is well to begin the treatment with raw milk or milk heated for a few moments. After one or two days, provided there is no diarrhea, diluted lemon or orange juice with a little sugar is permitted, and then the vegetables are given, so that the child receives alternately a small quantity of stewed fruits or vegetables, at first once a day, later for two meals of the day.

While, for many children who have up to this time had only fluid nourishment, it is necessary to insist upon mixed food, nevertheless, great care must be taken to avoid dyspepsia. Particularly when there is vomiting it is necessary to decide whether the unusual food is the cause, or whether there is a beginning gastric dyspepsia.

If at the time of the change in diet diarrhea is present or appears soon afterward, it is well to dilute the raw milk with gruel which previously has been heated. Of the vegetables, mashed potatoes and the juice of elderberries will perhaps be best tolerated. It is not advisable to postpone antiscorbutic treatment until a time when the intestinal catarrh will have been cured, aside from the fact that an irritation of the colon may itself be due to the scurvy. Fixation of the diseased extremities, so long as they are sensitive, is to be avoided, and only in the very rare case of marked deviation at the point of fracture is it indicated later. While the pain continues all unnecessary movement of the child, even bathing, is to be avoided.

No remedy is known that will cure or even favorably influence infantile scurvy. This applies particularly to the preparations of iron and phosphorus. The swollen gums may be painted with tincture of myrrh. During convalescence there is no objection to the employment of drugs (for example, preparations of iron), but I have never employed such remedies, as a suitable nutrition and the hygiene of the skin, together with plenty of fresh air, have always been considered as more essential.

As was mentioned at the beginning of this article, the recognition of infantile scurvy must result in its disappearance. In the nutrition of infants all excesses in the heating of milk must be avoided, as well as the free substitution of milk by artificial preparations. Further, so soon as the teeth appear mixed food is imperative. Although in the treatment of infantile scurvy raw milk is advised, for the healthy child milk moderately heated is preferable. If we wish to maintain the principle introduced by the Soxhlet apparatus, the milk may be Pasteurized for half an hour at a temperature of 60° to 65° C. (140° to 149° F.)<sup>1</sup> or it may be boiled slightly over an open fire and then poured hot into clean bottles which are protected by Flügge's glass covers or an aluminum cap; but the cooling should always be rapid and the milk must be kept

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<sup>1</sup> For example, in Kobrak's apparatus.

cold. After the sixth month mixed feeding should begin in the manner previously indicated. How this is best to be extended (eggs and the like) does not come within the scope of this article. With a mixed diet the daily quantity of milk or milk broth may be limited to one or one and a quarter liters. That this form of nutrition is advantageous to the child has been proven satisfactorily by experience. [Offensive discharges do not contraindicate the immediate use of orange juice, on the contrary. Decoctions of oatmeal or barley when added to the raw milk act as anti-fermentatives. Open windows, day and night, and cold air stimulate a healthy metabolism. The long list of foods recommended above is a rather long one for a baby less than a year old.—EDITOR.]



# RICKETS (RACHITIS)

BY J. ZAPPERT, VIENNA

## I. INTRODUCTION. HISTORY

*Rickets is an anomalous constitutional disturbance of early childhood, characterized by alterations in the growth of the skeleton and recognized by them.*

The term *rachitis* was introduced by Glisson, who probably discovered a relationship therewith of the common curvature of the vertebral column (*ῥάχις*, vertebral column), but by his use of the single letter r, instead of rh he indicated the English popular designation of the affection, *rickets*. German thoroughness, for philologic reasons, decided the use of the word *rhachitis*, but it is not necessary to out-Herod Herod and we have now generally accepted the popular spelling with a simple r.

The common German designation for the affection is "the English disease" (first described by English authors) or "double limbs," that is, double growth, so-called because of the enlargement of the epiphyses. The French designate the affection "*rachitisme*," by which, perhaps, they express in a better manner than by our doubtful term *rachitis* the constitutional nature of the affection rather than an inflammatory process.

Whether the affection was known to the civilized peoples of antiquity is questionable; the quotations in this respect from ancient authors are as little convincing as a hump-backed statue of Aesop of the classical period, frequently quoted, which much more likely represents recovery from spondylitis. Nevertheless, it is remarkable that Soranus, in the second century A. D., mentioned the occurrence of curved legs in Roman city children as being more frequent than among the country population—a statement which might very well be utilized in favor of *rachitis*. The finding of probable rachitic curved bones in Grecian graves has been regarded as evidence of the prevalence of the affection in ancient times (Epstein).

Still more uncertain is our knowledge relative to the presence of the disease in the Middle Ages, although we are not justified in drawing conclusions from the lack of reports during that period of mental apathy. Of interest in several respects is a recent report of Stegmann before the Vienna Anthropological Society that rachitic, curved bones have been found in the graves of South American natives which antedate the time of Columbus. A remark of Theodosius in the year 1584 (Stöltzner) certainly has reference to a rickety child.

These and a few isolated early reports of physicians do not alter the fact that medical knowledge of the affection dates from the year 1650, at which time an English physician, Glisson, published his monograph upon rickets, (*de rachitide sive morbo puerili*) which has since become celebrated. The cumulative appearance of this affection in English districts resulted in a medical commission for the study of the affection, the outcome of which was Glisson's report. This author gave such a comprehensive description of the affection that to him belongs the credit of the first professional observation of the disease, notwithstanding some preceding and simultaneous reports of other writers.

The literature of rickets dates back to about 1840. New reports followed in constantly closer sequence which treated of the entire symptom-complex of the disease or of certain portions of it, and in particular its etiology, pathology, and treatment. At the present time the reports are so numerous as to exceed those of almost every other affection of infancy. Recent advance in the knowledge of the disease is associated, among others, with the names of Elsässer, Virchow, Ritter v. Rittershain, Kassowitz, Pommer, Spillmann, and Stöltzner. The last author recently published a book on rickets with a compilation of about 450 reports—perhaps the most noteworthy work upon the subject in the last two centuries—without absolutely exhausting the subject.

In spite of this extraordinary example of industry, intelligence, observation, and reflection we are to-day but little further advanced than we were fifty years ago in our knowledge of "the English disease." Rickets has become an actual battle-ground for specialists in children's diseases. Scarcely a disputed question in the great realm has been solved; pathogenesis, anatomy, treatment, and even many points in the symptomatology are yet the subjects of active and often exciting controversy. When it is seen how results which were regarded as positively proven are constantly combated with new arguments; how theories, the tenableness of which was declared undoubted, have been disproved by newer observations; how investigators, who believed that they had raised a monument by their researches in this direction, have been overthrown by opponents, we must admit that a great amount of labor and thought have been devoted to this theme, but we cannot escape the impression that often personal factors—rigid adherence to what has once been stated, inherent opposition to views set forth by one or the other side—have played a great rôle in the discussion of rickets and have sometimes prevented the objective opinions from assuming the importance due them.

It is to be hoped that a later generation of physicians will succeed in clearing this well-cultivated but bewildering field of labor, and bring to a *finale* this extraordinarily interesting chapter of rickets.

## II. DISTRIBUTION, FREQUENCY, INFLUENCE OF SEASON

### I. DISTRIBUTION

There has long been a definite knowledge of the extraordinary distribution of rickets in all countries of the temperate zone (Hirsch). The only uncertainty related to its occurrence in northern countries, upon certain islands, in highly mountainous regions, and in the tropics; but a careful investigation in such localities has yielded positive and increasing results. Thus Johannessen found numerous cases in Norway (Christiania), Feer met with the disease in the high valleys of Switzerland, Kassowitz in the Tyrol; Fede found as many rickety children in Naples as in Northern Italy; in Sicily (Palermo) there is an institution for rachitics. Monti diagnosticated rickets in Japanese children, four years of age, who had immigrated into Germany; Stöltzner found the disease in mulatto children (in Berlin); Palgrave saw it in Arabia; Moncorvo found numerous cases in Rio de Janeiro, among the blacks as well as in the white population. Under these circumstances it is a question whether the reports in regard to the immunity of certain regions, which are often based on general observation, especially of children who can creep and walk, will not require correction after an exact study of infants and the adoption of the usual diagnostic measures. Some thoroughly competent observers, however, have stated that Iceland, the Färöe and Ionian islands, Roumania, Turkey, Algiers, Mexico, Peru, China, Japan, India, Java, Canada, etc., may be regarded as free from rickets, that is, regions in which few cases occur.

That *racial peculiarities* also play a rôle, as is generally assumed, has not been positively proven. It is true that the Caucasian race in general shows a particularly marked predisposition, but since investigations in this direction have demonstrated the presence of rickets in negroes and in Mongolians, it is questionable whether an accurate research will not show merely a simulated preference for the white race, that is, that a residence in large cities explains the condition. The racial influence and the cumulative effect which are shown among the immigrant Italians of North America (Snow) may also have their explanation in the great poverty which exists among this portion of the population. The immunity from the English disease which is maintained among Gipsies, Kirghiz, and Kabyles demands an investigation of their offspring.

Notwithstanding these skeptical remarks the possible and even *probable immunity from rickets in certain races and countries* is by no means to be denied. From the importance which is given, perhaps justly, to heredity (see below) it might readily be thought that in some people under favorable circumstances (mode of living, residence in the open air, etc.) the appearance of rickets could be prevented; that by enforced surroundings (islands, mountains) or social relations (Gipsies, Turks, etc.) an inbreeding may have transmitted this immunity further, so that even to-day these races have almost no rachitic children. Perhaps rickets will be a valuable example in the highly



interesting problem of the importance for the health of the people of inbreeding and mixed marriages.

## II. FREQUENCY

Somewhat more uniform are the views concerning the *frequency* of rickets in the regions in which it exists. Kassowitz calculates that 89 per cent. of the children of his out-patient service in Vienna are rachitics. Epstein gives about the same figures for Prague and Kissel for Moscow. Jouconsky calculates about 90 per cent. for St. Petersburg (Baumel), while another author mentions only 32 per cent. for the same city. Mey found 80 per cent. in Riga. In *London*, according to Baumel, 92 per cent. of artificially-fed children under one year are rachitic. In Berlin, according to Cohn, 65 per cent., according to Hauchecorne and Stöltzner, 90 to 95 per cent. In German cities, among other reports there are those of Munich (Seitz), 72 per cent., Frankfort (Rehn), 50 to 60 per cent. Of the French reports those of Chaumier (Tours) show 50 per cent., of Baumel (Montpellier), to whom we are indebted for a comprehensive compilation of these figures, 50 to 60 per cent., while Marfan calculates about one-third of the population of Paris to be rachitic. In Basel Feer has estimated 86 per cent., Johannessen in Christiania 66 per cent., Fede in Naples 50 per cent., and Moncorvo in Rio de Janeiro 50 to 80 per cent.

From these figures it appears that the English disease is very common in various cities of Europe. The variations in the above figures preclude a positive deduction as to the unequal frequency, for the figures of some authors are taken entirely from hospitals, others cover only definite periods. Further, many dispensaries (for instance that of Kassowitz in Vienna) are especially frequented by rachitic children; therefore Mey's idea to include only vaccinated cases appears advisable. Some of these differences in the figures may be due also to the various diagnostic view-points, whereby one investigator may assume rickets where another will note nothing abnormal. Finally, the question of frequency, as well as of the distribution of the disease, rests largely upon the exactness with which the clinical material is studied. The admitted errors of certain authors are very instructive. Thus, Fischl, in the reports of the Munich Children's Clinic mentioned only 5.1 per cent. of rachitic cases, whereas Seitz, by a careful investigation of the same clinical material (children under one year), was able to find 72 per cent. Baumel calculated the number of rachitics in Montpellier to be 4 per cent. but was obliged to increase the figure to over 50 per cent. when the question was more closely studied for the purpose of a paper before the Moscow Congress. A similar error was experienced by Feer in Basel. Lange reports that in the same region of Saxony one physician had estimated 2 per cent., another 50 per cent. of rickety children under one year of age. These figures show conclusively how the accuracy of the reports regarding the distribution and frequency of the disease is dependent upon the precision of physicians who have made a specialty of children's diseases, and how little the approximate figures and general conclusions signify.

Rickets occurs principally in the *crowded quarters of large towns*. It is more common among children of cities than of the country; but even in the affluent classes it is by no means rare. Kassowitz found rickets in 89 per cent. of his *charity patients* and 59 per cent. among his prosperous clientele, but the well-to-do classes are more exempt from the severe forms of the affection, and particularly from the continuance of the disease after the second year of life—due unquestionably to favorable hygienic conditions.

Whether *climate* is an influential factor in rickets is doubtful. Hagen-Torn has attempted to prove by careful examination that the affection is confined to regions of great humidity (80 per cent.), but Shukowsky has quite properly suggested that in moist climates children are confined to the house more than in dry regions, therefore moisture has only a mediate influence upon the appearance of rickets.

On the other hand it is unquestionable that *absence of air and light and the lack of an open air life exert an unfavorable influence* upon an existing predisposition, i. e., upon the course of the affection. This may explain the relative rarity of rickets among the rural population in the warmer climates and high valleys with powerful winter sun (Davos), and the cure of rachitic patients in the country, at the seaside, or in the mountains. Keller maintains that in houses which are shaded he has found more cases of rickets than in those exposed to sunlight (Feer). From this, however, it must not be concluded that the “mountain air,” as such, gives immunity from rickets, as Feer was able to demonstrate the affection comparatively often in manufacturing districts of dense population and high altitude, while secluded villages at the same elevation showed very few cases. The fact that people who have immigrated from the plains into high valleys bring up more rachitic children than the endogenous population (Feer) also gives rise to thought and enjoins us not to overlook the importance of hereditary immunity in the subject of favorable climatic conditions.

### III. SEASON

An important argument for the unfavorable influence of indoor life, as demonstrated by Kassowitz, is the *season curve* of rickets.

According to several reports (Klein-Schwechten, Wallach, Alexander, Quisling, Feer, Mey, etc.) it is evident that the number of rachitics who flood the dispensary in children's hospitals in the early months of the year (February, March, April) is much greater than in the summer and in the autumn. The rachitic curve, therefore, has a rapid ascent in the spring and a descent in the summer.

The cause of this rachitic curve is questionable. Kassowitz bases upon it his “respiratory theory,” according to which the deleterious effects of winter residence in poorly ventilated rooms is said to be the actual cause of rickets during this season. But Lange quite properly objects to this, for the reason that the ascent of the frequency curve of rickets in the spring in the out-patient service may also be due to other causes—associated bronchitis; possi-

bly because they are more in the open air. We will content ourselves with the fact that Kassowitz' explanation of the rachitic curve is true of the ambulatory patients of the hospitals of large cities, without seeking for further conclusions.

It would be of more interest to know whether, as Seitz believes, more rachitic children are *born* in the spring than in the autumn, or whether the reports of Bystrow and Pavone (quoted by Monti) that children born in the autumn are more frequently attacked by rickets than those born in the summer, could be confirmed by investigation in children of the prosperous classes. At present this theoretically important question is unsettled.

### III. AGE OF ONSET. CONGENITAL RICKETS

*Is rickets a congenital disease?* The answer to this question is of cardinal importance for the pathogenesis of the affection. All theories relative to disturbances of metabolism, respiratory and other deleterious factors are of secondary consideration and, in a part of the cases at least, would be transferred from the child to the mother if it is true that rickets is a congenital disease. Unfortunately there are vast differences of opinion concerning this which make a decision difficult.

The marked anomalies of the skeleton which are now and then found in the new-born, and which are designated *fetal rickets*, must be left out of discussion. These congenital deformities have nothing in common with rickets and had better be designated by another term (*osteogenesis imperfecta* or *osteoporosis congenita*, *chondro-dystrophia* or *fetal myxedema*) than to confound the condition by intimating a relation to rickets.

*The arguments in favor of actual congenital rickets* may be covered by three questions: 1. Are there in the new-born distinct *clinical* evidences of rickets? 2. Are the rachitic diseases of the bones of the new-born to be considered *histologically* rachitic? 3. Does a case of undoubted rickets develop from the rachitic symptoms of the new-born?

1. The occurrence in the new-born of clinical symptoms, seemingly rachitic, has been demonstrated by a number of investigators. Such symptoms are craniotabes, large fontanelles, and the rosary. After the expressed views of older authors in this direction (Elsässer, Bednar, v. Ritter) Kassowitz demonstrated rickets in premature infants and in the children born at term. Unruh confirmed this and assumed the congenital nature of all cases of rickets. Schwarz, in Vienna, found nearly 80 per cent. of 500 infants, born in the Vienna Lying-in Hospital, to be rachitic. Feyerabend, in Königsberg, estimates the number to be about 70 per cent. Quisling, in Christiania, whose statistics as to the frequency of rickets are below the average, was able to demonstrate 11.5 per cent. rachitic cases among 200 new-born. Feer reported 63 cases of rickets among 100 new-born in the Basel Women's Clinic. H. Neumann is also an adherent to the theory of congenital rickets, while Heubner is a strong



opponent. Some authors call attention to the high rachitic figures for the first weeks of life, which might be considered to indicate an intrauterine onset of the affection (M. Cohn, Kassowitz). Therefore, in regard to the *clinical symptoms* there is, with but few exceptions, a unanimous opinion as to the occurrence of congenital rickets. After comprehensive investigations I have reached a similar conclusion, but would attach greater diagnostic value to the softness of the occiput than to other anomalies of the skeleton.

2. Anatomical researches relative to *congenital* rickets are few. After the individual observations of Virchow, Mori, and Bornträger, Kassowitz, upon the basis of histologic examinations of the bones of new-born infants and of children of premature birth, firmly maintained that rachitic changes could very frequently be demonstrated (in 26 among 29 cases). A number of subsequent investigators, as Fede, Lenz, Tschistowitsch, and more recently Escher, have failed to confirm these lesions. The last-named author was unable to find any clinical symptoms of rickets in 105 children examined by him, since he denies that flexibility of the cranial bones at the margin of the sutures and distinct prominence of the epiphyseal boundary of the ribs are rachitic phenomena. Had he thought otherwise he would, like Schwarz and others, have found 80 per cent. of his patients rachitic. Kassowitz's view is confirmed by Fischl, who proved histologically that the prominence upon the cartilage of the ribs in the new-born were the signs of rickets.

Here apparently there is an insurmountable contrast between adherents and opponents of the intra-uterine origin of rickets, which depends entirely upon the interpretation of anatomical specimens.

Our knowledge does not justify a decision as to whether Kassowitz's findings or those of his opponents are the correct ones. In any event the *stage of onset* of rickets is exceedingly obscure, and anatomical researches, which might illumine the subject, are limited. Our decision becomes all the more difficult if only the bones of the skull are examined, the softness of which is conclusive in the diagnosis of congenital rickets. The uncertainty of the findings in the histologic preparation is made obvious by a remark of Fischl to the effect that Tschistowitsch's findings, which are interpreted as being opposed to congenital rickets, "completely coincide with the histologic picture which Spillman outlines of the initial stage of the disease."

3. Light is shed upon this obscure realm by a recent work of Spietschka in Prague. After M. Cohn had demonstrated that from insignificant rachitic symptoms in the new-born distinct rickets arises later, Spietschka again investigated the subject in a large number of cases in Epstein's Foundling Asylum at Prague, with very decisive results:

*In all those children, who, a few days old, were transferred from the Maternity Department to the Foundling Hospital with symptoms which were manifestly rachitic, the symptoms increased during several months of observation to well developed rickets, which was only controlled by treatment with phosphorus. The percentage of children born with rickets is estimated by Spietschka to be 50 per cent. (in 1,468 cases).*

The author arrives at the following conclusions: "The softness of the occiput (craniotabes), the so-called defective ossification of the skull, the gaping sutures and their flexible margins, the disproportion between the circumference of the head and of the thorax in the *new-born child*, yield at once to that transformation which in the other child is generally recognized as the typical rachitic skull. As a rule, when these cranial phenomena appear in the new-born the remainder of the skeleton undergoes a rapid change. These peculiarities of the skull in the new-born child therefore represent the first symptoms of rickets. Accordingly *there is a congenital rachitis. Most cases of rachitis are congenital.*"

There can scarcely be any objection to this method of proof provided we do not wish to transfer the diagnosis of rickets from the sick-bed to the post mortem room. Fischl quite properly emphasizes that for a diagnosis of rickets in the new-born not only the bony changes but the entire habitus, especially the distended abdomen, require consideration.

If, after what has been stated, the pros and cons of the intra-uterine development of rickets are weighed, we must admit that the clinical evidence on both sides is so overwhelming that, so long as the onset of the rachitic bony changes is not absolutely clear, the question cannot be decided by pathological anatomy alone.

*Nevertheless, the occurrence of congenital rickets is very probable, and the possibility must be considered that every case of rickets arises from a congenital basis, and that external circumstances probably favor the development of rachitic symptoms, but do not produce them.* Unruh, Feer, and Spietschka are more or less acknowledged adherents of this view, although the last two, from their statistics, only maintain that rickets "usually develops intra-uterine." It will scarcely be possible to solve this puzzle by means of figures. We will always find infants in whom, on account of its insidious development, no clinical symptoms of rickets can be demonstrated, and so long as the disease of the bone is the decisive diagnostic criterion it will be impossible to decide whether or not a constitutional predisposition or a dyscrasia prevailed before the appearance of the skeletal anomalies. Finally, analogous to certain positive congenital nervous diseases which do not appear until later in life, we cannot deny a congenital predisposition to rickets beyond the age of infancy. However, we will not pursue this purely hypothetical theme further, as a too great generalization might destroy the foundation of those cases of rickets in which the intra-uterine origin appears to be certain.

*On the contrary, according to clinical experience, we must maintain that, in addition to congenital, there is an acquired form of rickets in which the symptoms develop under observation.*

Reports concerning the age of greatest frequency vary according to the views of different authors as to the intra-uterine onset of rickets.

Baginsky, an opponent of congenital rickets, never saw the affection in children younger than three months, and considers the period of acme to be between the twelfth and eighteenth months. Guerin and others name similar

ages. Anders maintains that florid rickets belongs to the second year of life (Mey). In contrast to this Ritter remarks that rickets is most frequent in the first year of life, and that then one-third of the cases are observed during the first six months. Kassowitz also has noted the majority of the cases in children under one year. The following instructive table is given by M. Cohn:

0- 6 months	.....	62.8	per	cent.	rachitic,
7-12    "	.....	66	"	"	"
1-1½ years	.....	72.1	"	"	"
1½-2    "	.....	70.6	"	"	"
2-3     "	.....	61.6	"	"	"

From this it is seen that the frequency of rickets gradually increases in the first years of life and that *the disease is actually most common in children aged one year or a year and a half*. Contradictory reports like those of Baginsky, who saw no case in children under three months, and of M. Cohn, who in the first six months found rickets in 60 per cent. can only be explained by variations in the diagnostic view-points which permit one author to utilize changes to which the other attaches no significance.

*Rachitis tarda*, i. e., the appearance of florid rickets in children four or five years of age, has been described by many writers (Biedert, Gärtner, v. Genser, James, Duplay, Hutinel, Aucher, Knöpfelmacher, and others). In this category also those cases of typical rachitis must be included which had existed in early infancy and had been cured prior to a reappearance of the disease.

In how far curvature or flexibility of the bones at the *age of puberty* and later (flat-foot, etc.) have to do with rickets cannot be discussed here.

The rachitic bony changes of the skull, the pelvis, the extremities, etc., which are noted *in the adult* do not belong to this affection as such, but are the remains of a process which occurred in infancy, and may be designated rickets as little as *maculæ corneæ* may be termed a scrofulous inflammation of the eyes.

#### IV. HEREDITY

The fact that rachitis may often develop intra-uterine is not equivalent to heredity. *Congenital* rickets may be *acquired intra-uterine* but need not therefore be *inherited*. Also, from the colossal distribution of rachitis among the children of large cities, the conclusion may be drawn with some basis of certainty that the disease was widespread in the previous generation, and that, therefore, undoubtedly a descent of rachitic children from parents who had been rachitic can be statistically proven; but this cannot be utilized as a proof of undoubted heredity. It would still remain questionable, however, whether rachitic children are in fact the offspring of parents who have suffered from the same affection, and, if this is the case, whether external conditions have not brought about the development of the disease in the mother as well as in the child.



Undoubtedly these are the reasons which have led authors to omit from discussion this problem of rachitis, and to report only individual observations. Thus Feer found that mothers with rachitic residues, even when they subsequently dwell in regions free of rickets, bear children with the English disease. Elgood knew of a woman who had children by three husbands; only the children of the father who showed distinct remains of rickets were rachitic. Siebert, after some comprehensive studies, has lately approached the question somewhat more closely. He reported observations, covering several years, of families of which the mothers in some instances were affected by rickets and others were free of the disease. He found that as a rule all of the children of the former suffered from rickets, while the offspring of healthy mothers were usually exempt. He determined also that the manner of feeding of the children, as well as the usually unfavorable surroundings, had no decided influence upon the affection, although breast-feeding, while it did not prevent the appearance of rachitis in children hereditarily predisposed nevertheless excluded the severe forms. The period of breast-feeding is also without influence on the development of rickets. Siebert, with great certainty and consequence, maintains the heredity of rickets, upon which other factors are more or less dependent. His evidence is not convincing in all particulars, but his conclusions coincide with many practical experiences and force the problem of heredity in the English disease into the foreground.

At all events *the heredity of rickets, the congenital occurrence, and the racial immunity* unite in a triad of facts which, if their entity could be established, would be of the greatest significance in the pathogenesis of this affection.

## V. VIEWS REGARDING THE ETIOLOGY OF RACHITIS

Although in the foregoing the significance of heredity and of the congenital origin of rickets has been especially emphasized, this does not justify a rejection of the many *theories in explanation of the occurrence of the affection*. Neither of these factors have been positively demonstrated and a vast number of cases remain which must be regarded as acquired; furthermore some of the forebears of a rachitic generation must have contracted the malady. Finally, in the majority of these patients undoubtedly the acme of the disease is reached in the first year of life or later; therefore even the strictest adherent of heredity and of the congenital onset must assume unfavorable extra-uterine influences in the etiology.

As a matter of fact, the number of theories regarding rickets, and the labor expended upon them, is enormous, yet no one of them can be considered as at all adequate.

In general the hypotheses are based on the following conditions: *Insufficient lime metabolism and nutritive disturbances; acid intoxication; deleterious respiratory influences; infectious factors; anomalies of an internal secretion.*

I. Nutritive hypotheses: A. One of the oldest and perhaps most dis-

cussed theories of rickets is that which assumes as the cause of the affection a *damage to lime metabolism*.

a) As deficiency of lime in florid rachitic bones is unquestioned, it was first assumed that in children who become rachitic *too little* calcium was present. The main support for this theory was obtained by animal experiment, in which softness of the bones was produced through a deficient supply of lime. But examination of the milk of the nurses, both of rachitic and non-rachitic infants, showed that in either case the amount of lime is the same. It has also been demonstrated that cow's milk is much richer in calcium than human milk; nevertheless artificially nourished children are more liable to the English disease than breast-fed infants. Even if we take into account the precipitation of a part of the lime in sterilization, a surplus of calcium is still ingested by the child (Marfan). Only with permanent and exclusive feeding with infant foods (without milk) is a deficit in lime possible, but in most countries this form of nourishment, in contrast to feeding with cow's milk, does not play a practically important rôle. *A deficient ingestion of lime, therefore, cannot be considered in the pathogenesis of rickets.*

b) If, therefore, insufficient lime is not the cause of rickets, the adherents of the calcium theory conclude that *lime is not sufficiently absorbed*, so that the blood and consequently also the bones are deficient in calcium. This want of absorption is accounted for in several ways. Some authors (Seemann, Zander) regard a lack of hydrochloric acid in the stomach as the cause of insufficient solution and absorption of lime. This would also explain the frequent digestive disturbances in rachitic children. The apparent deficiency of hydrochloric acid, however, was not confirmed by the subsequent investigations of other authors (Bouchard, Labbé and Klecinsky). On the other hand, Zweifel assumed that the calcium assimilated in a soluble form becomes insoluble in the intestine and so leaves the structure—a view which was disproven by Soxhlet. We are indebted to Rüdel and Rey for the experiment which decided this question. Following Vierordt's initiative, and having previously determined a uniform calcium metabolism, these investigators gave to children who were rachitic, and also to those who were free of the affection, a single dose of chalk; both groups of cases reacted promptly and uniformly by an increased excretion of lime.

This experiment proved that *calcium is just as rapidly and readily absorbed in the English disease* as under normal conditions and, therefore, that the cause of rickets is not to be found here.

c) Finally, it was thought that while the lime was ingested and assimilated in sufficient quantity, an excess was excreted, and this was due to a surplus of acid in the blood which dissolves the lime in the bone and permits it to pass off from the body. This view of an *acid intoxication*, for which lactic acid particularly was held accountable (Monti),<sup>1</sup> may be positively abandoned, according to the experiments of Stöltzner alone and in conjunction with Bru-

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<sup>1</sup> C. Heitzmann.—EDITOR.

bacher. These investigators proved a normal alkalinity of the blood of the rachitic child, on the one hand, and on the other that the blood and the soft parts in rachitic subjects were not relatively rich in lime, as would be the case if the lime were absorbed from the bone.

It is true that some researches (Babeau) have proved it likely that in certain stages of rickets more calcium is excreted than under normal circumstances. This finds its explanation in the fact that calcium salts which have been separated from the bone pass off from the body; therefore we are dealing with a consequence, but not with the cause, of rickets (Fischl).

The main support for the lime theory was obtained from *animal experiments*. By feeding animals with fodder poor in lime, simultaneously with doses of lactic acid, a softness of the bones was produced (Rolloff, Baginsky, Voit). Many objections to the interpretation of these experiments were raised. This method probably gives rise to a general decrease in lime but not to a selective decalcification of the bones. Besides, the character of the osseous changes produced in the animal experiment is by no means histologically identical with the rachitic changes, but corresponds, according to Miwa and Stöltzner, to an osteoporosis which has nothing in common with rickets. Pfaundler has reached the same result by chemical experiments, namely, that the absorption of lime, introduced artificially into the tissues of animals through the vessels, increases the supply of calcium, which is not the case in rickets.

*Therefore, there is overwhelming experimental evidence to oppose the view of a relationship of lime metabolism with rickets*, and, although Zweifel has recently attempted to revive the theory by new arguments, it has been ultimately rejected by the majority of pediatricists.

*B.* The hypothesis of Delcourt, according to which potatoes and milk—the food of the poor—produce an excessive secretion of *potassium salts* in the body, thereby preventing sodium combinations in the bones, has found no confirmation among other authors (Wedensky, Spillmann).

*C.* Those views which regarded *digestive disturbances*, as such, i. e., the absorption of toxic substances from the intestine, as responsible for the development of rickets, were not much more successful. Spillmann, a prominent authority in the realm of rickets, has recently become an advocate of this hypothesis, but his single positive animal experiment (production of rickets by subcutaneous injection of the intestinal contents), when contrasted with the number of negative results, does not warrant such far-reaching conclusions. In any event, clinical experience is opposed to the theory, for certainly there are many rachitics who never have suffered from decided digestive disturbances, and accordingly the inclusion of intestinal disturbances among the initial symptoms of rickets becomes untenable.

Nevertheless, repeated experience, lately increased by Siegert's report, shows that there is a greater frequency of rickets, as well as a more severe affection, in artificially-fed infants than in those who are breast-fed, and the question remains whether unfavorable hygienic influences, poor nourishment, or a specific action of the food is the cause.



II. The influence of unhygienic conditions, especially unsanitary habitations, has suggested to Kassowitz the theory of *deleterious respiratory influences* for the pathogenesis of rickets. He believes that the life of children in unventilated rooms, the inhalation of foul air ("odor of the poor"), plays a dominant rôle in the etiology of rickets, and bases this view particularly upon the ascent of the rachitic curve after the winter months.

Although this theory includes some undisputable facts and claims a number of adherents, it is little more than a paraphrase of the statement that greater numbers of rickety children come under observation after the long confinement in rooms during the winter. Which of the deleterious agents that are common in the destitute population, as poor ventilation, improper food, lack of cleanliness, neglected disease, etc., is the principal factor is by no means obvious. The respiratory theory of rickets, therefore, is a figurative expression of an observation, but can scarcely be regarded as an etiologic hypothesis.

A more precise, scientific form was given to this theory by Wachsmuth, who assumes that in ill-ventilated rooms there is a surplus of carbonic acid which results in a surcharge of the blood. This excess of carbonic acid in the blood causes a greater solution of lime salts, and, by hindering the physiologic activity of the cartilage-cell, produces a splitting of lime in the bone. The deleterious product circulating in the blood also produces a contraction of the arteries and a consecutive hyperemia of the capillaries which prevent the cartilage-cell from reaching that stage of maturity consistent with its complete chemical activity. In consequence of digestive disorders there may also be a decomposition of the excess of lactic acid which leads to the production of carbonic acid in the intestine and its transmission thence into the blood. The result is then the same as in the respiratory excess of carbonic acid. While this theory of Wachsmuth's might satisfy our casual requirement, it will not bear close investigation. The occurrence of rickets in children who are well cared for and well nourished, its absence in cyanotic children with congenital disease of the heart (Edlefsen), and finally, the proof of normal alkalinity of the blood in rachitis (Stöltzner) have deprived the theory of its support, so that it remains only the hypothesis of a philosopher.

III. Rachitis has recently been included by some celebrated authors among *the infectious diseases* (Hagenbach-Burghardt, Edlefsen, Chaumier, Vierordt, Sterling). In support of this view the cumulation of rickets in certain streets and houses, its limited geographic distribution, the enlarged spleen, the analogy of the season curve with that of some infectious disease (pneumonia, polyarthritis), its preference for the osseous system whereby it resembles certain other infectious diseases of childhood, have been invoked. Nor did we have to wait long for the finding of bacteria in the bones of rachitics (Mireoli) which actually appeared to confirm this hypothesis. But active opponents have not been lacking and to some extent they are justified (Kassowitz, Fischl). The cumulative appearance of rickets in certain quarters of a large city, and its geographic distribution, find explanation in unhygienic conditions, racial peculiarities, and heredity (see above). The massing of the affection in cer-

tain houses is scarcely surprising, for we are dealing with the slums, and the children crowded into these districts have 80 per cent. of likelihood to contract the disease. The analogy of the season curve of rickets and other diseases has not been definitely proven; as explained above, the increase in the former does not necessarily indicate an actual frequency, and there is never an occasional cumulative appearance in other seasons, as is the case in other infections. Enlargement of the spleen is not a regular accompaniment of rickets (see below) and need not necessarily signify an infectious disease. Fever does not belong to the symptom-picture of rickets but is due to complications (see below). The analogy with other infectious diseases of the osseous system in infancy (syphilis, scrofulosis) loses its significance if we consider that, at this age particularly, the bones are the regions of active blood circulation and of growth, and if we further consider other non-infectious conditions of infancy which markedly involve the skeleton (myxedema, chondrodystrophia). Finally, according to Spillmann's subsequent comprehensive researches and experiments, the positive bacteriologic findings may be regarded as accidental contaminations.

Therefore, the etiology of rickets cannot be determined from these considerations, as it is impossible to decide whether arguments or counter-arguments are the more weighty. It would seem, however, that *the evidence in favor of an infectious or miasmatic origin is outweighed by proofs to the contrary.*

Oppenheimer's hypothesis that malaria always precedes rickets, and Parrot's theory of a preceding hereditary syphilis, are merely of historic interest.

IV. The most modern phase in the etiology of rickets is Stöltzner's theory of a *functional disturbance of an organ with internal secretion*. Other authors have held similar views: Thus, Knöpfelmacher and Heubner, who fed the thyroid gland to rachitic children, and Mettenheimer and Mendel, who fed the thymus gland. Stöltzner has included the suprarenal gland in the circle of his considerations and has attempted therapeutic researches. None of these procedures, however, has furnished convincing results and therefore cannot be utilized from the standpoint of treatment. Nevertheless, there is an encouraging thought in this idea which perhaps will have a beneficent influence in the question of the etiology of rickets. For example, the analogy with myxedema, which Stöltzner suggests, is noteworthy and might explain the congenital appearance, the hereditary influence, and the geographic and ethnographic distribution of rickets. It does not seem necessary to assume infectious-endemic causes to be responsible for the absence of function of the organs in question, as does Stöltzner, since they have not yet been proven for endemic cretinism. We might much more assume a *hereditary predisposition, perchance like the uric acid diathesis, wherein, as previously mentioned, the intermarriage of afflicted individuals furthers the appearance of the English disease in the descendants; that parents from a rachitic-free stock would generate an apparent immunity in their children and in the race.* It is true, external influences would also have to be operative to produce rickets in one predisposed child

while another is exempt. The question still remains, what factors are first active in families previously free of the disease.

In this sense, even if we assume the correctness of the last theory, the importance of nutrition and of the environment of the child would be worthy of further study as regards the pathogenesis of rickets.

## VI. SYMPTOMATOLOGY AND DIAGNOSIS

Some authors describe *prodromal symptoms* of rickets. By this they designate either disturbances in the respiratory and digestive organs, which are also regarded as the cause of the disease, or nervous irritability, sweating of the head, and sleeplessness.

I cannot agree with either of these views. Diseases of the respiratory and digestive tracts, as will be explained later, are not inevitable accompanying conditions, much less the precursors of the English disease; and the nervous manifestations, if due at all to rickets, are not prodromes but accompanying phenomena, the affection being already apparent by a softness of the occiput.

In a malady so eminently insidious as rickets *we can as little determine the time of onset as of cure*, but we must not forget that pathologic processes have existed in the body prior to the appearance of changes in the skeleton, although their recognition was at the time impossible.

*Symptoms actually characteristic of rickets, and not occurring in any other disease, are furnished by the osseous system alone*, the lesions of which must naturally occupy the greatest part of the discussion of the symptomatology.

First we shall describe those symptoms which might be regarded as the criteria of *congenital rickets*. The earliest lesions are found in the bones of the head. The sutures of the skull, especially the sagittal suture, are gaping, the edges of the frontal fontanelle and the sutures are soft and yielding, the posterior fontanelle is not completely ossified, upon the back of the head and in other portions of the skull there are soft, parchment-like areas. Sometimes the greater portion of the tabular part of the occipital bone is not firm in consistence and yields to pressure of the finger. Such a change of the skull is found in children who are normal at birth, as is shown by their habitus and weight, as well as in premature infants. Inversely, premature children may be born with a perfectly hard occiput. Besides these characteristic cranial phenomena of congenital rickets there are secondary osseous changes. The costochondral borders are thickened, but even normally there is a slight bead-like prominence, so that the transition from a normal to a pathologic condition is extremely difficult to define. Likewise, curvature of the tibia and the "rachitic habitus" of the new-born, made prominent by Fischl—distended abdomen, thick-set thorax with broad base—are not present in all cases of congenital rickets.

The symptomatology of "*acquired*" rickets, which arises after birth, is more distinct. The disease then shows itself most prominently in those portions of the skeleton which are undergoing the greatest development. This



gives to the sequence of rachitic symptoms a certain degree of regularity that varies according to the period of onset.

The greater number of cases of acquired rickets develop in the first half year (often in the first three months), with softness of the occiput which Elsässer has well defined as *craniotabes*. Notwithstanding the fact that its relation to the English disease was for some time in doubt, it is one of the earliest and most distinct symptoms of acquired as well as of congenital rickets. If the skull of the infant is examined in the recumbent posture, with the head grasped in both hands, palpation of the occiput in the region of the posterior fontanelle, at the occipital suture as well as in the dependent portions of the parietal bones, will reveal the presence of *craniotabes* in the form of a few soft areas which yield to the pressure of the finger like a fully distended bladder. This softness of the posterior skull, often only of the size of a cent or of a quarter dollar, is sometimes of such wide circumference that only the protuberantia occipitalis and its surroundings are uninvolved. The soft, yielding occiput becomes *flattened* by the constant recumbency of the child, and the hair upon this part of the head, which is scant even under normal circumstances, is almost worn off. A very common accompanying symptom of *craniotabes* is *sweating of the head*, particularly of the posterior portion, and after the child has been asleep for some hours the pillow may be saturated. Whether the softness of the occiput causes pain or disagreeable sensations is doubtful. It is true such children are frequently restless and sleepless, and a cotton pad as a support for the head appears to exert a soothing effect, but children who are models in sleep and in disposition are also affected with *craniotabes*. *Craniotabes* only occurs in those children who are kept mostly in the recumbent posture; therefore constant pressure of these growing bones against the underlying support is of causal import in this localization of rickets. Opposed to this is the occurrence of *craniotabes* in regions of the skull not exposed to pressure, as well as the fact that young dogs are also affected by *craniotabes*, notwithstanding the fact that in repose their head is supported upon their paws.

*Craniotabes* is a *common symptom of rickets and usually introduces the entire symptom-sequence* of the affection. It nearly always appears during the first half, rarely during the third quarter, of the first year, and sometimes continues after the second year; but Monti remarks, quite rightly, that *craniotabes* in these later years dates from a period prior to the sixth month of life, and that rickets occurring after the sixth month does not give rise to *craniotabes*. The frequency of the softness of the occiput is indicated by the following compilation of M. Cohn from the poor population of Berlin:

Of 100 children in the first three months of life 53.8 per cent. were affected with *craniotabes*; in the second three months 43.4 per cent., and in each of the last two quarters of the first year 38.35 per cent. These figures are certainly not too high.

The second symptom of rickets of the cranium is a *deficient involution of the frontal fontanelle and softness of the sutures*.

That the frontal fontanelle in a non-rachitic child gradually becomes smaller after birth <sup>1</sup> has been positively demonstrated after many controversies (Hochsinger, Kassowitz, M. Cohn). On the other hand, there is some doubt as to the time *when* a normal closure should occur. It is well to limit this period to the fifteenth or eighteenth month; not to the end of the first year as Kassowitz is inclined to do. This difference is important, as controversies may arise regarding the possible presence of rickets in children at the beginning of the second year. Often, however, the frontal fontanelle in rachitic children is still open at the age of two and three years, but then other symptoms of rickets are usually noted.

For comparison of the size of the frontal fontanelle in normal and rachitic infants, the following table of M. Cohn's is instructive:

AGE	AVERAGE SIZE OF THE FRONTAL FONTANELLE IN CUBIC CENTIMETERS	
	Normal Children	Rachitic Children
1-3 months	2.71	4.04
4-6 "	2.62	4.44
7-9 "	2.38	5.0
10-12 "	2.25	4.88
13-15 "	1.50	3.54
16-18 "	0.67	2.95
19-21 "	0.31	}
22-24 "	....	
2-2½ years	....	0.98
2½-3 "	....	0.55

*From this table it is evident that the frontal fontanelle of a rachitic child not only closes later but is larger than normal, and that in the first year of life it increases instead of decreases in size.*

In addition to enlargement of the frontal fontanelle in the rachitic skull *the surrounding bone is not hard and sharply marginated, but is soft and yielding*, so that in some cases the transition from the bone to the connective-tissue covering of the fontanelle is apparently gradual. This softness of the borders may also be recognized in the sutures of the skull, particularly the sagittal, the gaping of which in rachitic skulls frequently prevents an exact delimitation of the fontanelle. Kassowitz illustrates these fontanelles quite aptly by the figure ∞ (without end). In such skulls, with wide fontanelle and

<sup>1</sup> Many of our readers will not agree with the author when he says that "the frontal fontanelle in a non-rachitic child gradually becomes smaller after birth." He takes that to be "positively demonstrated," though he admits that a number of writers are of a different opinion. Constant observation of healthy babies will show that the large fontanelle grows up to the 7th or 8th month with, or rather in consequence of, the rapid lengthening of the lines of the adjoining bones.—EDITOR.

gaping sutures, it is not always easy to distinguish between a simple rachitic and a hydrocephalic cranium.

Anomalies in the size of the fontanelle and in the closure of the sutures, as the above table shows, appear, like craniotabes, at an early age, and are therefore a *primary symptom of the affection*.

A third characteristic abnormality of the skull in rickets is the *prominence of the frontal and parietal protuberances*. This is due to bony deposits in these regions and gives to the forehead a very conspicuous convexity ("Olympic brow"). If the top of the cranium is depressed on account of the retarded ossification of the fontanelle, and the occiput flattened in consequence of craniotabes, the skull assumes a rectangular shape which is quite aptly designated *caput quadratum*. The deposits of bone produce an increased circumference of the head which, however, does not indicate an increase in the capacity of the skull.

The thickenings at the protuberances of the frontal and parietal bones, previously mentioned, do not correspond to the most florid stage of rickets but are the signs of beginning consolidation. They are therefore most marked at the end of the first year and later, and remain permanently as rachitic changes of the skeleton. In the half-grown child, and also in the adult, the "rachitic cranium" remains an important witness of the English disease, although in a moderately developed skull the boundary between rickets and family peculiarity is not always easy to determine.

In severe rickets of the skull there are often *disturbances in growth of the facial bones*. The *superior maxillary* is elongated and narrowed, resulting in a higher palatine arch and a decided protrusion of the teeth. The *lower jaw* shows a characteristic change in that an angular projection at the point of contact of the anterior and lateral portions forces their approximation. This deformity of the lower jaw is of practical interest for the reason that the space for the development of the teeth is limited and thus their eruption and growth are hindered.

Of much greater and actually popular importance in rickets is the *cutting of the milk teeth*. The opinion, so often expressed, that rachitic children "teeth with difficulty" finds its support, on the one hand, in the nervous symptoms which are sometimes present, and on the other in the late and irregular eruption of the milk teeth. When rickets begins early the first teeth, instead of erupting in the sixth or eighth month, do not appear until toward the end of the first or in the beginning of the second year. If rickets begins late the first incisor teeth may appear at the proper time, after which dentition is arrested and the remaining teeth are not erupted until far beyond the normal period. This delayed dentition is one of the most frequent accompanying symptoms of rickets. There are also marked cases of rickets with normal dentition of the milk teeth. Inversely, a delayed appearance of the deciduous teeth must not be regarded as an absolute sign of rickets, as it may be the expression of a family predisposition.

Rickets affects not only the temporary but also the *permanent teeth*. The



abnormal formation of the jaws, mentioned above, forces the teeth together, causing deformity and a tendency to caries. The latter condition is further favored by the fact that the *teeth of rachitic individuals, particularly the incisors, are often poorly developed and distorted by fissures, indentations, and erosions*. This imperfection of the permanent teeth is due to the fact that the tooth pulp, during its development in the first years of life, is subject to the rachitic process like the osseous system. As the dentition of the incisor teeth and of the first molars occurs during the acme of rickets—in the first and second years—conspicuous changes are observed in these structures also. Therefore an incisor tooth, appearing in the sixth or seventh year and covered with erosions, is not a symptom of existing rickets but the result of a previous affection. The eroded incisors, which readily become carious, constitute one of the most conspicuous signs of rickets, although, as will be shown later, they cannot be employed in differential diagnosis in the absence of other phenomena. They represent permanent rachitic lesions which can be utilized beyond the age of infancy in the diagnosis of a former disease.

In addition to the skull, the *thorax* frequently undergoes marked changes. At an early age, during the existence of craniotabes, thickening may be noted at the costochondral articulations. When the thorax is palpated these thickenings give the impression of a chain of beaded prominences and therefore have obtained the designation "*rachitic rosary*." They are usually more marked in the lower ribs. They represent one of the most constant early symptoms of the disease, becoming distinct at the end of the first or beginning of the second year, or perhaps even in the congenital affection.

Rickets gives rise also to *other very conspicuous and serious changes* in the thorax. In consequence of the softness and inelasticity of this structure the ribs undergo a change in shape which is manifest by a depression of the upper lateral portion of the thorax and by a sharp indentation at the point where the ribs and their cartilages unite. An alteration of contour results whereby the lateral walls of the thorax, instead of being arched, are flat or even concave. The upper thoracic wall is protruded and rises almost to an angle with the lateral portion. If, with this, there is a flattening of the posterior wall, instead of showing the normal ellipsoid the thorax may have almost a square appearance. This malformation affects only the upper portion of the thorax. The lower portion bends outward so that the deeper ribs appear inverted. A narrow upper portion and an extremely wide lower portion of the thorax results, between which a depression is noted, particularly upon forced respiration, which corresponds to the lower border of the lungs—*Harrison's furrow* (Stöltzner, Vierordt).

This thoracic deformity may show various modifications or be altogether absent in rickets. At its extreme it presents that configuration known as "*chicken breast*" (*pectus carinatum*). Here the anterior portion of the ribs and the sternum are so prominent that the chest is wedge-shaped and its sterno-vertebral diameter in excess of the frontal diameter. Another not infrequent alteration of the thorax is produced when the costal cartilages at their insertion

with the sternum are bent backward and sink groove-like below the level of the anterior thoracic wall. In addition to the softness of the ribs this deformity of the thorax owes its origin largely to the respiratory muscle-tug of the diaphragm, the weakness of the other muscles of respiration, and the protruded abdomen (Vierordt).

The changes of the thorax are the forerunners of later rickets and are most distinct about the middle or at the end of the second year. Notwithstanding their conspicuousness they often disappear in late childhood, so that in the adult there are only indications, seldom a well-developed type, of the rachitic thorax. These marked deformities have a deteriorating influence upon the ventilation of the lungs. Even with an intact respiratory apparatus they often give rise to dyspnea and diseases of the respiratory organs which lead to frequent and dangerous complications. It is evident that in such diseases forced respiration exerts a deleterious influence upon the shape of the chest, so that cause and effect in rachitic disease of the thorax may form an unfortunate combination.

Children with rachitic thorax very frequently evince pain when they are raised by the axillæ—a symptom which may be referred to the bones, but might also be due to the fear of increased dyspnea.

Scarcely less common, but not associated with such serious consequences, is the effect of the rachitic process upon the *vertebral column*. This becomes unusually flexible, and when the children begin to sit up a *very marked kyphosis of the lower spine* develops. This curvature of the spinal column is usually transitory and rarely leads to permanent malformation, although it may be the cause of much anxiety to the parents. It belongs to the early symptoms of rickets. Of greater importance is the dextro-convex dorsal scoliosis, such as appears in rachitic children between the ages of two and five years. This often persists and produces compensatory curvatures of other portions of the vertebral column and permanent deformities of the chest. True scoliosis is not a very common symptom of the English disease. The curvatures of the spine which are observed in school-children and in adults are not always referable to early rickets; as a rule they are acquired later.

That in high-graded scoliosis displacements of the *pelvic girdle* must occur is in consonance with well-known suppositions. It is not these changes, however, which play the fatal rôle in rickets, but alterations in the shape of the flexible pelvis which develop early from pressure and weight and lead to permanent narrowness. The flat and usually narrow rachitic pelvis is well known to the obstetrician and is one of the most serious consequences of the English disease on account of the great obstacle which it may raise in labor. In contrast to most other rachitic anomalies of the skeleton, this change is of secondary importance in the florid stage of the disease and is of little diagnostic value, but in women it constitutes a dreaded permanent sequel.

*The shoulder girdle and the upper extremities* undergo various changes.

The *clavicle* is frequently thick, with the normal curvature greatly exaggerated. Not infrequently it is the site of infractions and fractures which are

often first recognized by decided callus formation. The distortions of the clavicle are usually combined with rickets of the thorax and appear at the same age. They are sometimes a permanent sequel of rickets.

Rachitis of the *shoulder-blade* produces a thickening and plumpness of the bones which may be recognized upon the spina scapulæ but are of no diagnostic importance.

In the *upper extremities* the *enlargement of the epiphyses of the wrists* must first be mentioned. Their circumference is often decidedly increased and they are distinctly separated from the carpal bones by a depression at the joint. From the two prominences which are thus formed upon the sides of this furrow the popular designation "double joint" has been derived. These epiphyseal protuberances appear with the first symptoms of the disease, sometimes appearing in the second six months, and add to craniotabes, wide fontanelles, and the rosary, one of the most valuable diagnostic criteria of rickets.

Curvature of the *humerus* may be demonstrated anatomically as well as clinically, and infractions are by no means rare.

The *bones of the fore-arm* often show a convexity upon the extensor side which is mostly referable to the muscle-tug of the flexors and pronators. Sometimes there is an actual spiral curvature of these bones which results in a permanent pronation of the hand (Vierordt). In the lower arm also infractions and curves may be noted. The curvature which appears in the lower third of the left fore-arm is attributed by Stone to the continual pressure at this point of the little finger of the person who leads the child.

Finally, in the *basal phalanges* of the hand a thickening may be found (Fischl, Neurath) which resembles a carious process or an osteochondritis luetica.

As these manifold changes, with their many combinations, lead in severe cases to very *conspicuous deformities, as well as to shortening of the arm*, they constitute an important diagnostic aid, although they cannot be employed much before the second or third year. Functional disturbances of movement may be associated with these changes whereby *frequent flaccidity of the joints and decreased muscular activity*, and possibly a painfulness of the bones, are subservient factors. These malformations of the humerus are relatively benign and tend to disappear, no matter how great their prominence in the acute stage of rickets. They are rarely seen in adults and when present are always associated with other gross changes of the skeleton.

Of greater importance and subsequent effect are the rachitic changes in the *lower extremities*. These represent the final link of the chain of rachitic anomalies of the skeleton and first appear after walking and standing have exerted their deleterious effect upon the bones of the legs, although mild grades of the disease, such as curvature of the tibia, are said to exist prior to these, and, it has been asserted, can even be recognized in congenital rickets.

The *epiphyseal protuberances of the legs* do not possess the same diagnostic value as those of the arms, although they are quite distinct in the ankles.



*Curvatures of the femur* are not rare and are very conspicuous. A special form of the disease involves the neck of the femur, which joins the diaphysis less abruptly than normal, so that instead of an angle of  $126^{\circ}$  to  $128^{\circ}$ , it forms one of  $100^{\circ}$  or less. This causes adduction and outward rotation of the leg—the so-called *coxa vara*—and therefore interferes with the gait.

The changes in the *lower leg*, especially in the *region of the knee*, are most important. Lateral or anterior convexities of the tibia and infractions are very common. According to the combination of these curvatures in both legs various postures result which Comby very aptly illustrates by the following signs: ( ), O-legs, genu varum (bow-legs); X, X-legs, genu valgum (knock-knees); D, unilateral, genu varum; K, unilateral, genu valgum. These by no means exhaust the possible positions of the legs.

The result of the abnormal shape of the knee-joint is a peculiar position of the *foot*, which is aggravated by the flaccidity present in the joints of the legs.

The origin of *flat-foot* may also be referred to rickets, but in half-grown individuals caution must be observed in attributing these static changes to a rachitic source.

The changes in the legs, particularly the severer grades, are not a regular accompaniment of rickets and need be feared only in a small minority of early cases. When once developed they remain for some years, but even then there is a tendency to compensation; therefore the advice of Veit, Kamps and others to desist from operative interference until the sixth year is well justified. Nevertheless, the number of cases is not small in which curvature of the legs is a permanent sequel and one of the most unfortunate consequences of the English disease. The bones of the leg like those of the arm are retarded in their longitudinal growth, and in severe rickets there is occasionally a shortening of the entire osseous structure—*rachitic dwarfism*—such as has become familiar to us from the old pictures of “court dwarfs.”

A *résumé* of the anomalies of the skeleton in acquired rickets, as regards their onset, frequency, and diagnostic importance, gives us the following:

In the *first six months* *craniotabes* is the most reliable diagnostic sign of rickets. In the *second half-year* may be added the *rosary*, *enlargement of the frontal fontanelle*, and *epiphyseal protuberance at the wrist*. Toward the end of the first or in the beginning of the second year the *incomplete closure of the fontanelle*, *delayed dentition*, and the *arc-like kyphosis of the lower dorsal vertebræ* gain in diagnostic importance. In almost every case of early rickets these phenomena are combined. Not only do they belong to the most common symptoms but they are also the most reliable factors in diagnosis.

In further sequence, in the *second and third years*—but only in severe forms of the disease—*deformities of the thorax and of the clavicle*, *curvatures of the vertebral column and of the extremities* are added, the most conspicuous of which are those of the legs, with the previously-mentioned anomalous positions of the knee which usually appear latest, often at the end of the third year. At this age, in the majority of cases, though not invariably, *craniotabes* has disappeared. The other primary changes, however, may continue, and the open

fontanelles may persist after the disappearance of all the late symptoms of the affection.

The disease may be arrested at any moment, between the development of craniotabes and that of curvature and shortening of the legs; therefore the number of rachitic cases with flattened occiput is considerably greater than the cases with curvature of the extremities. The severer grades of rickets usually occur in neglected children, that is, those in destitute circumstances; in the well-to-do a typical rachitis of the thorax and extremities is rare.

Inversely, rickets which is not manifest until the end of the first year may lack the initial symptoms, especially craniotabes, and at once present the later changes. It is characteristic of such cases that the first milk teeth appear at the usual time but that long intervals elapse in the further processes of dentition.

This combination of late symptoms is most prominent in that form of the affection designated *rachitis tarda*, in which the osseous changes begin after the fifth year and sometimes not until the seventh or eighth year. These children complain of lassitude, cease to walk, and upon examination usually show conspicuous malformations of the skull, thorax and extremities. Such cases are not always of recently developed rickets but are the remains of an incomplete recovery, that is, a revival of the affection which had existed in early childhood.

Conspicuous and disfiguring as these osseous changes may be in the florid disease, nevertheless only a minority of them terminate in lasting alterations of the skeleton, in *permanent sequels* of rickets. We expressly refer to *permanent sequels*, not to *permanent symptoms*, because the osseous deformities in the adult do not possess the importance of residues of disease but are merely the product of cure, even though associated with alterations in the normal shape of the skeleton. The majority of the milder cases recover without sequels. A moderately severe affection sometimes leaves the broad head with prominent forehead and parietal bones, improper arrangement and composition of the teeth, and also the narrowed pelvis—a scarcely obvious, although very important anomaly. Only in extreme cases do permanent convexities of the legs and deformities of the thorax and of the vertebral column occur, and especially rachitic dwarfism.

The fact that rachitic children show no evidence of the affection in adult life must serve as a warning that too far-reaching conclusions must not be drawn from the absence of rachitic changes in the parents in behalf of the disease in their children.

With the changes in the skeleton the *characteristic and most valuable diagnostic symptoms* of the affection are exhausted. The clinical as well as the anatomical lesions—as we shall learn later—which are found in *the viscera* in rachitic subjects are neither peculiar to this disease nor are they always encountered. Therefore they may be regarded less as *genuine factors* than as *accompanying symptoms* of rickets, and only from the frequency with which

the individual organic changes are found can we conclude that they are in intimate relation with this disease.

The *general nutrition* is affected in various ways. With a rapid development of disease of the bone the increase in weight is retarded and the general condition is unfavorable, but, in the first year of life particularly, rachitic symptoms are observed in children that are fat and apparently robust. The *pallor* which is conspicuous in such infants is in contrast to the seemingly good condition of nutrition, and is seldom absent in well-developed cases. There is no clear explanation of this pallor. Blood examinations by Schiff, Widowitz, and others have revealed nothing characteristic of the disease, and the report of v. Jaksch that the salts in the blood are apparently decreased is thus far an isolated finding.

*Fever* is never a symptom of rickets, and when present it is due to complications.

*Chronic eczema of the head* (impetigo, seborrhea) of marked grade is often associated with rickets.

Whether *enlargement of the spleen* belongs to the symptom-picture of the English disease is the subject of discussion (Kuttner, Starck, Fox, Ball, M. Cohn, Müller, etc.). The decision depends upon whether every palpable spleen is enlarged, and whether a splenic tumor is to be referred to rickets or to an accompanying affection. I agree with the majority of investigators that the occurrence of a palpably enlarged spleen in rickets is common though by no means constant, and believe we are justified in regarding it as an important accompanying symptom.

*Enlargement of the lymph glands* is always the sign of some complication (Fröhlich). This is also true of *hyperplasia of the thymus gland* (status thymicus).

The *digestive tract* usually shows conspicuous derangement in children suffering from rickets. First, the *distended abdomen* must be mentioned since it is usually regarded as the consequence of chronic digestive disturbances. This view, however, seems doubtful when the large abdomen is observed in the new-born or in children but a few weeks old, and when we consider that the characteristic frog-belly of the rachitic is generally absent in non-rachitic children even in chronic intestinal diseases. Therefore it is much more likely that this distention is due to a peculiar configuration of the abdomen, occurring only in the English disease, which may be the cause but not the consequence of disturbances of digestion.

Great importance is attached to *functional disturbances of intestinal digestion* in rickets, which are regarded by some authors (notably Vierordt and Monti) as characteristic or etiologically important. Anorexia often alternates with bulimia; great discharge of gas is associated with colic; evacuation of dyspeptic feces of acid reaction alternates irregularly with constipation and the discharge of hard yellow stools. Bouchard and Comby believe that dilatation of the stomach is a regular accompaniment of rickets. No one will deny that all of these manifold disturbances of digestion—with the exception, per-



haps, of dilated stomach—occur in rachitis. In how far, however, they are peculiar to the disease, that is, are influenced by it, is difficult to decide, as, in the first place, investigations into the nature and frequency of intestinal disturbances in non-rachitic children have not been instituted; secondly, as Elsässer was aware, rickets may appear and continue without conspicuous disturbances of digestion; finally, an aggregate appearance of the disease after severe intestinal catarrh has by no means been observed. Nor must it be forgotten that the theories of rickets which are based upon disturbed digestive chemism are not yet proven and that pathologic anatomy has not shown any important or characteristic intestinal lesion. Therefore *intestinal disturbance* in rickets has not the importance of a symptom but at most can only be ascribed to an accompanying condition wherein it is still uncertain whether rickets favors the appearance of digestive disturbances, that is, retards their cure.

*Enlargement of the liver* is not rare in rickets but does not always accompany enlargement of the spleen.

Much more serious are the accompanying symptoms and complications on the part of *the lungs*. As already mentioned, rickets of the thorax interferes with respiration, especially inspiration. This leads not only to dyspnea and to difficulty in cough but to insufficient ventilation of the lungs, to a ready development of catarrh, and to lobular pneumonia. These bronchial catarrhs are capable, through the forced respiratory activity, of increasing the deformity of the thorax and indeed of the entire skeleton. There is therefore a reciprocal action of rachitis of the thorax and diseases of the respiratory organs which certainly justifies the view that *chronic catarrh and severe pneumonia are direct complications of rickets*.

Of diagnostic importance is the fact, mentioned by Rilliet and Barthez, that the alteration in the thorax and thickening of the shoulder-blades may, in percussion and auscultation, simulate diseases of the thoracic organs. Thus, over the supraspinous fossæ occasionally there is absolute dulness; again, the cardiac dulness is sometimes apparently increased, or the respiratory murmur has, here and there, a pseudo-bronchial character. Stöltzner therefore quite properly suggests that in percussion of the rachitic thorax we should not enter upon "subtle refinements" but should rather depend upon auscultation.

Nevertheless, the occurrence of *cardiac hypertrophy*,<sup>1</sup> especially of the right ventricle, in some rachitic children cannot be denied, but it must be regarded as a consequence of the increased resistance in the lesser circulation, due to pulmonary disease, rather than as a sequel of rickets. The over-exertion of the heart is probably the cause of the increased pulse frequency observed in rachitics, to which attention was called by Rilliet and Barthez and by Guérin. Diseases of the respiratory organs, together with excessive cardiac activity, represent the

<sup>1</sup> The cardiac hypertrophy discussed by the author is often apparent only, and then due to the flattening of the (normally elliptic) chest which brings it into contact with a greater surface of the heart muscle.—EDITOR.

gravest consequences of rickets and are frequently the cause of death in children suffering from that disease.

The *urine* shows no special characteristics. Some authors mention a disagreeable ammoniacal odor from the diapers of rachitic children after thorough wetting.

Attention has several times been called to *muscular weakness*<sup>1</sup> in rickets—an important although little understood symptom of the affection. Here we are dealing with a disturbance of function which is very conspicuous and to the parents is often the first indication of the disease. The children are backward in raising the head, sitting up, and in learning to walk. The last function in particular is mastered slowly and painfully and there is a preference either to sit with legs crossed like an oriental idol or to creep. Delay in the function of walking is so common in the well-marked disease that it may be regarded as a valuable sign of recovery from rickets.

Also in rachitic children who have learned to walk there may be a sudden arrest of this function without an obvious increase of the symptoms. This has been designated by Comby *pseudo-paraplegia*, as the apparent paralysis of both legs does not correspond to a nervous lesion. Besides the effect of this muscular weakness on the locomotion of these children it also plays an ominous rôle in respiration by lessening the action of the respiratory muscles, the stress falling largely on the diaphragm. In pneumonic processes there is ready paresis of the muscles of respiration. Even the movements of the arms and hands in severe rickets may be affected by this muscular weakness. Much as we appear to be justified in regarding, like Vierordt, the muscular flaccidity as an essential symptom of rickets, but little change is to be found in the muscles by direct examination. During life there is merely a certain degree of emaciation and want of tonicity, without direct paralysis or even limited muscular atrophy. Anatomical investigation reveals general atrophy and insignificant changes in the muscles which are explained sufficiently by the disease which is the ultimate cause of death.

Therefore the question must constantly arise whether the flaccidity of the muscles is a substantive factor of rachitis or whether it is a *consequence of pain in the rachitic bones*. But even if we agree upon a sensitiveness of the bones, our view is not well founded. It is true that children with rickets of the thorax often cry when their chest is touched, that frequently the first signs of rachitis are those of great unrest, and that attempts to walk are at first

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<sup>1</sup> Muscular weakness is a very characteristic symptom when rachitis makes its appearance early. It is observed both in the voluntary and involuntary muscles; and a cause of constipation in apparently normal, but rather fat, bloated babies about the third month. Part of the large size of the abdomen is the result of this incompetency of the intestinal muscle. The appearance of constipation at *that time*, while there was no change in feeding, is (almost) pathognomonic for rickets, in the same way that the congenital constipation of breast-fed babies is attributable to an undue length and flexures of the sigmoid part. The above "intestinal disturbance in rachitis has *certainly* the importance of a symptom," and is not, as the author suggests, a merely "accompanying condition."—EDITOR.

resisted because of pain. On the other hand, older children do not complain of any disagreeable sensation in the bones and the anatomical examination of the skeleton, particularly of children who have evinced painfulness of the bones, is not different in its results from the examination of children in whom such conditions are absent. We may therefore consider a possible relation of muscular flaccidity and sensitiveness of the bones, but cannot decide the question finally.

A very interesting and much disputed chapter in the symptomatology of rickets is *its relation to a number of nervous symptoms*.

It is obvious that an affection which is concerned with the rapidly maturing organism after affecting the skeleton may also attack the central nervous system, for the development of this part of the body is particularly active in the first years of life. Kassowitz has investigated the subject and upon the basis of rich clinical experience has described an entire series of nervous phenomena which result from, or, more properly, are the symptoms of rachitis. These in the order of their frequency are: (a) sleeplessness, sweating of the head, fright from impressions of sight and hearing; (b) facial phenomenon (Chvostek's sign); (c) inspiratory and expiratory spasm of the glottis; (d) general convulsions; (e) hyperidrosis universalis; (f) Trousseau's phenomenon; (g) spontaneous attacks of tetany; (h) spasmus nutans.

It is quite impossible to relate in detail the many reports of the last decades concerning most of these nervous conditions of infancy, or even to indicate them. In the following we shall only consider the relation of these phenomena to rickets, and sketch briefly, in so far as that is possible, the views of the majority of authors regarding them.

The nervous rachitic phenomena mentioned by Kassowitz may readily be reduced to a few pathologic types or symptom-groups. These are *increased secretion of sweat and nervous unrest, spasm of the glottis, tetany, convulsions, and spasmus nutans*.

That *sweating of the head and insomnia* belong to the most frequent accompanying symptoms of early rickets, that is, to craniotabes, certainly cannot be denied. As previously stated they are sometimes considered as prodromes of the affection. *General sweating* is a less frequent, but nevertheless common, complaint. That in these symptoms rickets represents a causal factor is very likely, whether rickets of the skull is responsible for sweating of the head and sleeplessness, or whether these symptoms are also an expression of a general rachitic dyscrasia, as is the case in craniotabes.

*Spasm of the glottis* occurs during inspiration and expiration. In the former, after a few crowing inspirations, respiration is suddenly arrested and the respiratory organs remain fixed in that position. Inversely, in expiratory apnea (Kassowitz) the larynx and thorax are fixed in expiration. These phenomena occur in crying but may also be spontaneous. They usually last a fractional part of a minute and are associated with fright. In severe attacks, particularly when there is expiratory dyspnea, there may be cyanosis, loss of consciousness, and convulsions. Even in these cases recovery is frequent;



nevertheless the number of children who succumb to this spasm of the glottis is not small. Therefore, laryngospasm must be reckoned among the most dangerous diseases of infancy.

*Children who suffer from spasm of the glottis are mostly rachitic.* Whether or not laryngospasm is always due to rickets was the subject of an active controversy between Loos and Escherich on one side and Kassowitz on the other, which was ventilated at various congresses and in several publications, and on either side has gained many adherents among pediatricists.

In support of his view of the undoubted relation of rickets and laryngospasm Kassowitz has mentioned a number of facts: In the first place spasm of the glottis is found only in children with rachitic phenomena; further, the most frequent occurrence of both conditions is in the spring months; again, the prompt action of phosphorus in spasm of the glottis is a proof of its rachitic foundation. He has also given an anatomical explanation for the development of laryngospasm in his assumption that through the hyperemia of the rachitic bones of the skull certain cortical centers are irritated which give rise in the animal experiment to inspiratory and expiratory positions of the larynx.

In the critical analysis of these arguments *laryngospasm and tetany* cannot be separated. Although it must be admitted that in the individual case spasm of the glottis may occur without other symptoms of tetany, nevertheless their frequent combination together with the electric hyperirritability of the nervous system which occurs alike in both diseases (Thiemich) shows that as a matter of fact there is no distinction between laryngospasm and tetany. We cannot enter into all the details of this greatly discussed question of tetany, but in so far as its relation to rickets is concerned it is certain that *similar conditions are operative for spasm of the glottis and for tetany.*

Much may be said against a strict connection between rachitis on the one hand and laryngospasm and tetany on the other.

Both conditions are also observed in non-rachitic children, but then the opinion that an open fontanelle at fifteen months is a sign of rickets cannot be upheld. There is no congruity between the severe forms of rickets, especially craniotabes and laryngospasm, and tetany. On the contrary, nervous symptoms are sometimes absent in extreme cases of rickets and again may be present in the mildest forms of the affection. The coincidence of spasm of the glottis and tetany with an increased rachitic frequency in the early months of the year has its analogon in the appearance of tetany in adults during the same season. Besides, there are characteristic curves at different seasons for other nervous affections. Tetany and with it spasm of the glottis occur in certain years and in special regions in the form of epidemics, while in other countries (France) they are very rare. Rickets, on the contrary, is of about uniform frequency and is widely distributed even in regions free from tetany. At the time of onset of tetany or of laryngospasm the rachitic symptoms undergo no change and the process continues after the nervous phenomena have disappeared as though they had never existed. A change in the food may be fol-

lowed by a sudden disappearance of the spasm of the glottis or of tetany, where a rapid change in the rachitic disposition cannot be assumed. The relation of definite cortical centers to spasm of the glottis is very unlikely, for isolated spasm of the laryngeal muscles is never observed in other cerebral affections (tumor, meningitis, encephalitis), as would be inevitable if a cortical center were involved. Nor is it manifest why, of all the known centers upon the surface of the brain, only two, which are otherwise inactive, can be stimulated by hyperemia of the rachitic cranial vault, while *isolated* spasms of the extremities, of the ocular muscles, or of the facial nerve are absent in rachitis. In addition to this is the fact that particularly the laryngeal centers in question correspond to the frontal brain, which is therefore but little affected by the rachitic process, while functional anomalies in other centers, for instance in the occipital lobe, may be absent notwithstanding high-graded craniotabes.

Finally, the fact must be considered that tetany, with its constant bilateral spasms, with its increased nervous and muscular excitability, with its undisturbed sensorium, and with its absence of all cerebral symptoms, by no means corresponds to our conception of a cerebral clinical picture but probably has its seat in spinal centers or in the peripheral nervous system.

Therefore, the only remaining indication of the rachitic origin of laryngospasm and of tetany is the prompt success of treatment with phosphorus. But by no means does this appear to be reliable evidence, for it does not stand to reason that the result of phosphorus treatment, which is often noted within two or three days, should affect the osseous structure alone, in which weeks are required for the distinct appearance of changes. It is more probable that the phosphorus acts upon the nervous system the same as upon the bones, so that no conclusions in favor of a relationship of rickets and laryngospasm can be drawn from the therapeutic reaction.

The answer to these questions must be that we are not justified to include spasm of the glottis and tetany among the *symptoms* of rickets, and, although clinically there is a frequent coincidence of both conditions, the theoretic conclusions of Kassowitz must be rejected. Escherich, the former opponent of Kassowitz, has attempted to overcome this by advocating a subdivision of tetany under the term "tetany of rickets," to indicate that it mostly affects children suffering from the English disease but is not its consequence. But this cannot be regarded as a very happy expedient, for no decision is reached, and Escherich must also include in this group those cases of infantile tetany in which a typical tetany attacks a non-rachitic child.

This by no means decides the question of the relation between rickets and laryngospasm or tetany. New points of view are probably necessary to re-awaken an interest in the condition in the scientific world. A paper which Pineles recently read in the "*Wiener Gesellschaft der Aerzte*" seems to open a new perspective. According to this investigator tetany may be referred to a damage of the epithelial bodies in the vicinity of the thyroid gland. If we consider that Stöltzner refers rickets to a disturbance of an organ yet unknown with "an internal secretion," there is in this parallelism a possible

starting-point for investigations which may throw light upon the subject of rickets and its relation to tetany.

Similar to the problem of tetany in rickets is also that of *convulsions*. The frequency of general convulsions in rickety children must be admitted, but there is no absolute evidence that rickets exerts more than a predisposing influence on the readily active cause of convulsions. Therefore children with high fever and with intestinal intoxication are more prone to convulsions if they are rachitic, but thus far there is no reason to regard rickets, as such, as a causal factor. In these cases treatment with phosphorus by no means shows such brilliant results as in spasm of the glottis.

Among the nervous symptoms as yet unmentioned, the slightest relation to rickets is probably found in *spasmus nutans* and *nystagmus*. These constantly combined conditions also probably occur most frequently in the early months of the year, but are by no means dependent on the presence of rickets, and sometimes befall strong children who begin to walk early. Their duration is a few weeks, sometimes months. Whether treatment with phosphorus decidedly shortens the affection is difficult to prove.

Among the other nervous symptoms ascribed to rickets the following must be mentioned:

The *facial phenomenon* (*Chvostek's sign*), which is also encountered without tetany in rachitic children, has nothing in common with rickets, as it occurs in older children also in the spring months, and is not unusual in adults.

Disturbances of the *sense of taste*, which Lichtenstein claims to have noted in a great number of rachitics, frequently have not been found in subsequent examinations and cannot always be regarded as signs of simple rachitis (Büsem, H. Neumann).

Peculiar *cataleptic conditions*, in which older rachitic children remain for some time in unusual passive positions, have been described by Epstein and Thiemich. The symptom is not common and its relation to rickets has not been proven.

That there cannot be an absolutely characteristic *mental and intellectual* disposition of rachitics is evident from the fact that nearly 80 per cent. of the population of a large city were rachitic in their youth. In how far *restlessness and the tendency to scream* of many rachitics can be referred to a predisposition to disease, to stress of respiration, to sensitiveness of the bones, is difficult to decide. It is certain that in addition to these "bad" rachitic children we find others who are particularly friendly, gentle, and amiable.

Nevertheless, *it is an undeniable fact that many rickety children develop slowly, and especially are backward in learning to talk, which, together with tardiness in locomotion, give the impression of backwardness.*

It is likely that this mental tardiness does not belong to rickets, as such, but represents one of the most curious complications of the affection, namely, *hydrocephalus*. As to the frequency of rachitic hydrocephalus there are great differences of opinion (for example, Hensch and v. Ritter). There can be no doubt, however, from the clinical and anatomical findings that hydrocephalus



occurs not rarely in connection with rickets. It is true, the clinical picture of hydrocephalus is not very marked. Unusually great circumference of the head, open fontanelles and sutures leave a doubt in the individual case as to what part of the condition is due to rickets and what part to hydrocephalus. In rachitic hydrocephalus the symptoms of brain pressure and paralysis are absent, as well as a progressive or a regressive course which otherwise is peculiar to hydrocephalus. If in rickets the well-known hydrocephalic symptoms occur which relate to the position of the eyes, spasm, convulsions, etc., we are probably justified to search for other causes of hydrocephalus and no longer to regard them as purely rachitic. The only symptoms which characterize the hydrocephalus of rickets are headache, a certain degree of mental sluggishness, and retarded development of speech. It is possible that rachitic hydrocephalus predisposes to later *epilepsy*, although the importance of the "rachitico-hydrocephalic skull" mentioned so frequently in the history of patients with nervous disease must not be exaggerated.<sup>1</sup>

Of as little prominence as the clinical symptoms of rachitic hydrocephalus is its anatomical picture, in that we usually find nothing more than a moderate dilatation of the cerebral ventricles without obvious signs of a past inflammation. In fact this finding is not rare in rickets and must probably be regarded as symptomatic of the affection. How this accumulation of fluid in the cerebral ventricles arises has not been positively proven. Stöltzner has offered the following hypothesis: The periosteal proliferations of the cranium produce a narrowing of the communication between its internal and external spaces and cause a stasis of lymph in the interior of the skull which is manifested by dilatation of the cerebral ventricles. Stöltzner regards this theory as purely speculative, but it deserves consideration, since not an actual "rachitic disease" of the brain is assumed, but an occasional occurrence of rachitic hydrocephalus from disease of the bones.

A condition altogether obscure is the occasional finding in the autopsy of rachitics of *hypertrophy of the brain*, which is evident not only by the conspicuous size, but also by a special firmness of the organ. Clinically this condition gives rise to no distinct symptoms<sup>2</sup> and is scarcely to be identified as actual hypertrophy of the brain with symptoms of cerebral pressure which causes death (Schick).

This exhausts the most important points in the symptomatology of rickets.

*In what relation are these individual signs of disease with the rachitic process?* Which of them are conspicuous *symptoms* of the affection, which are

<sup>1</sup> In connection with hypertrophy it must strike us that previous to a final hydrocephalus there is œdema. These brains are always œdematous, and their functions influenced by the amount and locations of the parenchymatous œdema.—EDITOR.

<sup>2</sup> Hypertrophy of the brain *does* "give rise to distinct symptoms," if a large size of the head, with normally developed cranial bones and symptoms of pressure, with tendency to occasional convulsions belong to the class of distinct symptoms. The cases are rare, but discussed as early as 1857 in the *New York Journal of Medicine* ("On the premature ossification of the cranial sutures and fontanelles").—EDITOR.

the *direct consequences*, and which are frequent but not absolutely certain *accompanying conditions*?

Only the *changes in the skeleton* can be regarded as positive *symptoms* of the English disease. These, in their individuality and grouping, are found in no other disease and represent absolutely positive diagnostic criteria. Further signs of the rachitic dyscrasia are the *peculiar pallor and softness of the skin, the flaccidity of the muscles, the enlarged spleen, and the nervous unrest*. Whether the *digestive disturbances* are expressions of the rachitic disease or accompanying phenomena cannot be positively determined.

The primary *direct consequences of the rachitic osseous process* are diseases of the *respiratory organs*, and next in order those of the *circulatory system*. Here hydrocephalus as well as the questionable *painfulness of the bones* must be included, which, in combination with the muscular flaccidity, may cause *delay in the locomotor functions*.

A frequent *accompanying condition*, but by no means due to rickets, is found in the other nervous complications. Perhaps their coexistence is to be explained, as some authors assume, by the presence of etiologic factors common to both diseases.

We must now briefly discuss *the relation of rickets to certain other affections*.

After the rejection of Parrot's view, that every case of rickets is due to *hereditary syphilis*, the relationship of the two diseases is limited to the fact that children who are hereditarily syphilitic are especially subject to rickets. Recently Hochsinger has thoroughly investigated this question and has sought to prove it by figures, but his statistics show conspicuously that the rickets of syphilitics is of a particularly benign nature and is rapidly cured. If to this we add the reports of authors who have not noted a frequent occurrence of rickets in children with inherited syphilis (Stöltzner), the intimate relation of the two conditions becomes very insignificant and perhaps may resolve itself into the fact that debilitated children acquire rickets more readily than those who are healthy.

The theory of a causal connection of rickets with *malaria*, which Oppenheimer attempted to prove, has been entirely rejected.

The relation of rickets to *Barlow-Möller's disease* (infantile scurvy) is still the subject of discussion. The affection has been directly designated as *acute or hemorrhagic rachitis*. Their affiliation is limited to the common occurrence of Barlow's disease in rickety children—a circumstance which, as there are exceptions to it (Rehn), probably only represents the statistical sequence of the frequency of rickets. Artificial nutrition may possibly be a connecting link between rickets and infantile scurvy.

That rachitic individuals readily *incline to tuberculosis* has been known since the investigations of v. Ritter. Nevertheless, we must not consider their combination in the sense of a direct relation between the rachitic and tuberculous predispositions of the entire organism, but must regard the debilitated

constitution of rachitic children, the great tendency to diseases of the respiratory organs, and the common disturbance of the digestive faculty as the fundamental conditions for the successful entrance of tubercle bacilli.

It cannot be denied that in severe rickets, particularly when associated with hydrocephalus, there is a tendency to *epilepsy*, or, more correctly, that among adult epileptics there are conspicuously many with rachitic skulls. Here also there is an apparent necessity for a connecting link between rickets and the nervous affection, in which sense perhaps we may consider rachitic hydrocephalus. As we have found this affection to be in intimate relation to rickets we must necessarily designate epilepsy as a malady to which cases of recovered rickets show a predisposition.

## VII. DIFFERENTIAL DIAGNOSIS

The *differential diagnosis* of rickets presents no confusion if we adhere strictly to the changes in the bones, which, in their characteristic form and intimate connection, are not duplicated in any other disease. Affections of infancy which run their course with changes in the skeleton are most readily confused with rickets. Primarily among these is infantile *myxedema*, which often resembles the English disease in the enlargement of the epiphyses and the cartilage of the ribs, the delayed closure of the fontanelles, and the appearance of the teeth; but the peculiar physiognomy, the macroglossia, the nature of the subcutaneous cellular tissue and the hair, and the absence of sweating are unmistakable signs of the myxedematous disease, and even the bony changes, notwithstanding their similarity, have no affinity with those of rachitis.

*Hereditary syphilis* may in some instances be confounded with rickets. This would happen if a luetic osteochondritis at the joint ends were to resemble the rachitic enlargement, if luetic and rachitic hydrocephalus must be differentiated, if syphilitic softening of the bones or deposits upon the skull were present, if the distinction between rachitic and late luetic curvature of the tibia must be made, and if, finally, conclusions as to the presence of syphilis or rickets must be drawn from the changes in the teeth.

From these osseous symptoms of hereditary syphilis osteochondritis can, as a rule, be determined without difficulty by its usually unsymmetrical localization, by the pain, by the commonly accompanying paralysis (pseudo-paralysis luetica), and by the prompt relief after antisiphilitic treatment. Syphilitic bony deposits on the skull and craniotabetic softenings (Stöltzner) may cause great perplexity and can only be distinguished from rickets through the different localization of thickening, and by their sharper limitation and higher protuberance. On the other hand it scarcely seems possible positively to recognize a rachitic-luetic hydrocephalus in its late stages by the form of the skull, as the *cranium natiforme*, which was formerly supposed to belong only to syphilis, may occur also in rickets. In this case the differential diagnosis will often depend upon the clinical hydrocephalic symptoms, which



appear earlier in syphilis than in rickets. The curvatures of the tibia of syphilis tarda have the shape of a curved scabbard, in that they are flattened and are convex anteriorly, while in rickets the curvature is in the frontal plane. A rule, it must be admitted, with many exceptions! In the teeth we must remember that only the semilunar curvature of the two central upper incisors (Hutchinson's teeth) warrants a suspicion of syphilis; all other erosions and indentations may occur in other affections of infancy and particularly in rickets.

*Barlow's disease* may in its casual aspect be confounded with rickets and is often mistaken for it (acute rickets). The similarity exists in the painfulness of the bones as well as in the protuberances, though the former is more acute than in rickets and is mostly limited to the legs, and the latter is more marked in the diaphyses, which are much more frequently the seat of periosteal hemorrhage than the epiphyses. If, in addition, there is extravasation of blood in the mouth and the mucous membranes the diagnosis of morbus Barlowii can scarcely be doubted.

The permanent changes in the rachitic skeleton are differentiated from *osteomalacia* less by the osseous softening than by the development of the latter affection in the adult and the occurrence of the softening at a period when the rachitic changes would have become prominent and fixed. The occurrence of true osteomalacia in older children, in spite of reports to the contrary (Siegert, Roon), has not yet been determined with such certainty as to be of practical importance in a differentio-diagnostic respect. The difference between osteomalacia and rickets is found particularly in the anatomical relations and at this time is merely of theoretic interest. Stöltzner emphatically declares that the erection of these sharp anatomical divisions is unjustified.

Among the rachitic symptoms *kyphosis* may be confused with *spondylitis*, *rachitic pseudo-paraplegia* with *poliomyelitis*, the *distended abdomen* with *meteorism*, i. e., *tuberculous peritonitis*, *coxa vara* with *congenital luxation of the hip-joint*, and great difficulty may arise if the condition is not correctly understood.

*Radioscopy*, which has been so successful in the last few years, promises valuable results in the diagnosis of rickets. The reports which have thus far been published relate particularly to the rachitic child. While in a *normal* tubular bone, for example, on the distal ends of the radius and ulna or on the middle bones of the hand, the epiphyseal joint is sharply margined, as though lined with a ruler, and shows a decided linear shadow, in the *rachitic* bone the epiphyseal border is but slightly prominent, not in a straight line but as if beaker-shaped and fibrillated. Differences may also be recognized in the structure of the diaphysis, in that the parallel striations and regular contour of the normal bone make way for an irregular deposit of osseous trabeculae, and lighter areas are noted in parts that are but slightly calcified.

Differentio-diagnostic X-ray examinations in luetic osteochondritis (Hochsinger), myxedema, and mongolism have already been instituted, but thus far they lack systematic comparison.

## VIII. PROGNOSIS

*If we adhere strictly to the changes in the skeleton it must be admitted that rickets unquestionably terminates in recovery. In fact, we may doubt whether we are dealing with a disease in the ordinary sense but rather with a disturbance in development.* The bony changes which we recognize as the expression of "florid" rickets disappear in every case and the affection is never found in adults. Our attention, therefore, is not directed to a cure, as such, but to the prevention of too great a distribution of osseous changes and of deformity in the healed bones. The physician's object in this is similar to that in fractures, which, as is well known, may unite without treatment, but nevertheless require management to prevent a subsequent disturbance in function.

*In this broader sense the prognosis of rachitis is uncertain and not always favorable, as even treatment will not positively insure an arrest of the rachitic process, and in severe cases we are helpless against a recovery with permanent anomalies (for example, changes in the pelvis, kyphoscoliosis).*

If, in addition, we include those conditions which, although not directly belonging to the affection, are intimately associated with it, the prognosis becomes even less favorable and our treatment more inadequate. Thus the prognosis of the pulmonary affection in rachitic children especially is very much more serious than in healthy ones; the prognosis in laryngospasm, in tetany, and in convulsions, notwithstanding the favorable effect of phosphorus, is doubtful, and the cure of intestinal catarrh is mostly very protracted.

*In pulmonary inflammation and spasm of the glottis lie the main dangers of the rachitic. These complications represent causes of death which are to be regarded as the direct consequences of the English disease.*

Therefore, when there is any implication of the respiratory organs, or any other affection of infancy, rickets in general is a prognostically unfavorable factor, and the course of influenza, measles, whooping-cough, diphtheria, etc., in rachitic children is liable to be more severe than in children previously healthy.

But even "cured" rickets may have serious consequences for the person affected. Thus, a kyphoscoliosis upon the activity of the heart and lungs, a narrowed pelvis upon the course of labor, hydrocephalus upon mental development, general curvature of the bones and dwarfism upon social position and capability for various occupations. If we continue this conception, the emphysematous hump-back perishes, hemorrhage follows rupture of the uterus in consequence of narrowed pelvis, individuals suffering from rachitic deformities grow tired of life, and with decided narrowing of the pelvis death of the second generation may occur *intra partum* or the health may be permanently destroyed (hemorrhage of the brain, paralysis).

From this it is sufficiently evident that rickets, no matter how we regard the nosological position of the florid osseous changes, is a condition of serious import, and that professional activity, even though limited to the prevention of

too great a distribution and dangerous complications, has before it a wide and beneficent realm.

## IX. PATHOLOGIC ANATOMY

In the description of the pathologic anatomy of rachitis we are concerned almost exclusively with the characteristic *changes of the skeleton*. In this chapter we shall depend largely on the reports of Virchow, Kassowitz, Pommer, and Stöltzner, who have materially broadened our knowledge of the condition.

According to Pommer the rachitic disease of the skeleton is distributed over the entire osseous system and becomes especially manifest in those areas of most active growth of bone. From the anatomical standpoint the course of the affection may be divided into a *stage of onset*, of *acme*, and of *cure*, the transitions of which necessarily are not sharply defined. First, however, it would be well, for an understanding of the anatomical foundation of the affection, to describe the changes which take place at the acme of the disease.

The following must be emphasized as characteristic factors of the rachitic bony process: *hyperemia of the bones; irregular formation and proliferation of the tissues in which normally osseous formation occurs; deficient secretion of calcium in these tissues, and the pathologic decalcification of bones that already contain calcium*.

The normal production of bone occurs in the *periosteum* and at the *osteochondral borders*, and here the essential anatomical factors of rachitis are established.

The *periosteum* is for the most part decidedly thickened. It is closely adherent to the bone and can be loosened only with difficulty. The thickening is due to a hyperemic layer between the fibrous periosteal cover and the bone, consisting of alternate porous, plexus-like tissue and coarse longitudinal bands. Microscopic investigation reveals a substratum, rich in cells, which is interrupted by medullary vascular spaces. This "osteoid" tissue resembles the bony tissue, but differs from it in its reaction to stains as well as by the absence of the ordinary deposits of lime. It is true, upon the external layers of this proliferated periosteal cover there are disseminated deposits of calcium salts which gradually become richer as they invade the bone; but even in those areas in which ossification occurs the osseous trabeculae are surrounded by osteoid tissue. There is, therefore, a thickening of the periosteal layer with the formation of osteoid tissue, numerous blood-vessels, and a retarded ossification.

Even more conspicuous are the changes at the *osteochondral borders* of the tubular bones.

Section of a *normally growing bone* reveals between the osseous and cartilaginous structures a limited and sharply-defined bluish zone of calcification several millimeters in thickness. Histologically the bone formation appears about as follows: Cutting centerward from the cartilaginous epiphysis we



find that the cartilage cells, which were at first deposited irregularly, arrange themselves in definite rows corresponding to the direction of growth, and begin to enlarge. The intermediary substance of these cell columns takes up lime salts and then becomes the zone of provisional calcification. From points of vascular ossification in these layers of cartilaginous proliferation capillaries originate which cause resorption of the cartilaginous tissue and form medullary spaces, in the walls of which, by proliferation of cells introduced through the vessels (osteoblasts), true bony tissue forms and soon becomes a compact structure by deposition of calcium salts.

In well-developed *rachitic bones* these conditions are essentially changed. The osteochondral boundary is markedly widened—to 1 cm. in thickness—and conspicuously red. In contrast to the normal cartilage the end is not sharp but serrated. As in the normal bone, the cartilaginous cells are first arranged in rows and afterwards clump together, forming larger nests. The enlargement of the cartilage cells, however, is much greater. The lines of direction are less conspicuous the closer we approach to the diaphysis. The proliferating capillary branches permeate this “chondroid” tissue much more deeply than in the normal bone, so that in place of a uniform approximation of the cartilage columns and medullary spaces there is a quite irregular interlacing of these histologic elements. Of essential importance in rickets is the fact that through the action of osteoblasts a structure arises which resembles bony tissue; this osteoid tissue, however, as was seen in the periosteum, differs micro-chemically from true decalcified bony tissue and shows but slight tendency to calcification.

Therefore, in the enchondral ossification, just as in the periosteal, there is an *enlargement of the zone of transformation, a marked hyperemia of the same, as well as the formation of osteoid tissue and delayed calcification*. The last factor is of essential importance in rickets. There is a *conspicuous disproportion between the broad transitional zone*, which is ready for ossification, and the *slight deposit of calcium* which is found there. The consequence of this is an abnormal softness at the epiphysis and a ready separation of the epiphysis from the diaphysis in the macerated bone.

A further rachitic stigma is the *decalcification of bones already compact*. It is questionable whether this is an actual occurrence. Virchow has pointed out as an undoubted differential factor between rickets and osteomalacia that in the latter affection solid bone is absorbed, while in rickets the soft bony tissue does not become compact. This view was opposed by v. Recklinghausen and more or less by Pommer, Cohnheim, and Stöltzner. Heubner limits the resorption of bone to the severest “osteomalacic” forms of rickets.

Stöltzner has called attention to a change with loss of calcium of the bone, an *osteoporosis*. “A portion of the Haversian canal is often dilated, forming larger medullary spaces;” in many cases half or even more of the entire skeleton “is replaced by a loose, fibrous, medullary tissue.” Such an osteoporosis, which is found especially in the external portions of the corticalis, according to Schwalbe is normally present in children from the sixth month

to the end of the second year. It is Stöltzner's opinion, however, that it is more marked in rickets and is due to the fact that the apposition of the bone is far behind its absorption. An interesting result of Stöltzner's experiments in feeding animals with food poor in calcium was the occurrence of an osteoporosis of this sort without a rachitic change of the bone. This author also assumes that by treatment with phosphorus only the osteoporosis is influenced, the bones becoming firmer, but that the actual rachitic change—the deficient calcification of the osteoid tissue—is uninfluenced.

A review of the consequences which necessarily arise from these histologic changes in the *shape of the rachitic bones* gives the following: The bones are markedly hyperemic, alike in the periosteum, in the zones of ossification, and in the marrow. Through the great proliferation and the deficient calcification of the periosteum, as well as from the absorption of bone, they are abnormally soft. It is not difficult to cut or even to sever them, nor to change their shape by external force. This gives rise to the many deformities and curvatures of the skeleton with which we have become familiar from the symptomatology. In addition there are infractions and fractures which affect the shape of the bones of the extremities and the clavicle. A further consequence which has been mentioned is the softness and protuberance of the osteochondral borders of the long bones. Finally, there is an irregular periosteal deposit and by softening processes in the flat bones of the skull an irregular thickening and thinning is produced, the borders of the bone being thickened, the thinness affecting principally the squama of the occipital bone and the dependent portions of the parietal bones, in which probably unfavorable conditions are brought about by opposing pressure of the brain and of the underlying surface.

*In this series of bony changes all of those anomalies of the skeleton may be included which are met with in rickets:* Craniotabes, double joints, the rosary, narrowed pelvis, and finally, the curvatures and deformities of the thorax and extremities.

*How are these anatomical findings to be explained?* Two diametrically opposed views have been given: Kassowitz's law of a chronic inflammation and Pommer-Heubner's view of a primary deficient calcification.

Kassowitz regards proliferation and dilatation of the vessels as the essential characteristics of rickets. Deposition of calcium salts predisposes to sluggishness of the circulation remote from the centers of great blood supply. As there is an unusually profuse formation of blood-vessels, and therefore an active circulation at the points of ossification, the deposit of calcium salts is interrupted and the cartilage remains soft. An analogy for this deficient lime formation, due to over-active circulation of the blood, is found in large tumors and other tissues in which formation of calcium and even bone formation takes place in areas of deficient circulation.

To this theory of inflammation, which undoubtedly is an ingenious interpretation of actual findings, Pommer and Heubner are decidedly opposed. They did not find the histologic signs of inflammation, especially the small-cell infiltration, and regard the vascular proliferation as a secondary symptom.

On the contrary, that primary and essential in the rachitic process is *the formation of an osteoid tissue which possesses no tendency to prompt ossification.*

In connection with this Heubner attempts to construe a histologic picture of the zone of calcification that would arise if a rapid change of osteoid into bony tissue were to occur at the osteochondral boundary. Through the absence of the firm hold which is normally presented by the deposits of layers of calcium salts, the columns of cartilage cells as well as the newer capillaries lose their alignment and the ordinary growth of both tissues side by side is disturbed. Therefore the transition into bony tissue of the cartilaginous cells which have reached a certain degree of maturity fails to occur and the cell columns continue their growth and become unusually broad and long. On the other hand, on the diaphyseal side vessels have arisen, have formed medullary spaces, and have come in contact with the cartilaginous cell columns without the occurrence of uniform fusion. This theoretically constructed histologic picture actually corresponds with the findings in rickets. Heubner is therefore inclined to regard the deficient transformation of the osteoid tissue into bony tissue as the only determining factor, and does not recognize the "proliferation" of the cartilage, as such, but sees in this distribution of the cartilaginous zone a secondary factor of growth.

Having thoroughly considered the changes in well-marked rachitic bones, it yet remains to describe the *stage of onset and conclusion of the process.*

In regard to the *initial stage of rickets* there are not many reports and they are by no means unanimous. Spillmann describes as of primary moment the permeation of the vascular twigs into the cartilaginous tissue. These vascular twigs disarrange the cartilaginous lamellæ and cause their proliferation. The characteristic factor of the first rachitic change, therefore, according to Spillmann, is the irregularity of the line of ossification, due to the permeation of the vascular twigs. He attributes to the vessels the important initial symptoms of the rachitic osseous changes, and it is therefore self-evident that—in contrast to Heubner—he adheres to the view of an inflammatory process. Not only in the explanation but also in the recognition of the early rachitic changes there are many contrasts which attain some importance when the question of congenital rickets is invoked. In this respect the anatomical findings of craniotabes—the first symptom of rachitis—require comprehensive study.

The changes which the skeleton shows after *healing* of the process are better understood. After the zone of proliferation in the osteoid tissue is finally impregnated with lime salts and has undergone ossification, the bone becomes thicker, harder, distorted, and its surface beset with rough edges and osteophytes. This condition is especially distinct in the tubular bones, which lose their graceful contour, become heavier, and sometimes retain the shape which was produced in the florid stage by curvatures and inflections. Aggregations also occur in the cranial bones which increase the antero-posterior diameter. The influence upon the permanent teeth, the pelvis and the longitudinal growth of the tubular bones has already been mentioned.



A very interesting question, although at this time obscure, is *whether the organs of the body, as well as the skeleton, are affected in a characteristic manner by the rachitic process*. This pertains especially to the brain, the spleen, the liver, and perhaps the muscles; the frequent pathologico-anatomical findings in the lungs and in the heart are of a secondary nature, and in the digestive tract no regular form of affection is noted. It is true the anatomical findings in the organs mentioned are not well-defined, and are by no means characteristic of rickets, but the relatively common occurrence of hydrocephalus and enlargement of the spleen and of the liver, as well as the rare hypertrophy of the brain, in connection with the rachitic fundamental process cannot at once be ignored, although the assumption of Dickinson that these and other organs of rachitics show a peculiar hypertrophy of the connective and epithelial tissues scarcely corresponds with the actual facts. Vierordt's pathological findings in the musculature of the body, as already mentioned, are also scarcely characteristic of rickets.

Nevertheless, as Pfaundler has recently remarked, the possibility of visceral anomalies in rachitis in addition to the skeletal changes should not be abandoned. Stöltzner quite properly asserts that for this knowledge we should search less for morphological than for micro-chemical changes, i. e., by the process of staining. This method may furnish absolute proof of our view that rickets is a general disease, its manifestations but not its nature consisting of the changes in the skeleton.

## X. TREATMENT

The question of *prophylaxis* can only be considered in those cases which are to be regarded as *acquired* rickets. If we accept the view of a congenital rachitis as well as the influence of heredity, the prophylaxis must cover a much broader scope and must not only influence the health of candidates for marriage and of pregnant women but in every case of rachitis we should endeavor to confer immunity from rickets to the coming generation. In practice these considerations are superfluous and we must attempt to protect the child from all deleterious influences under which rickets flourishes, no matter whether we admit the acquirement of the disease or an aggravation of congenital symptoms.

The first attention must be devoted to *nutrition and plentiful light and air*. Even in the treatment of children already affected these factors are predominant, therefore *prophylaxis and dietetic treatment mostly go hand in hand*.

*Proper nourishment* of the infant has always been considered an important prophylactic factor in rickets and must so be maintained even though the theoretic basis of this requirement has not yet been established.

That good breast-milk fulfills this necessity of proper nourishment requires no proof, and its effect in weakening the hereditary predisposition to the disease has recently been proven by Siegert. But not all breast-milk is proper

food and we must agree with Vierordt, who regards artificial nutrition as far preferable to the milk of poorly qualified wet-nurses, who are perhaps changed several times. Since even the best breast-milk does not protect from rickets, we will formulate our requirement of proper nourishment in general by placing good breast-milk first, but that artificial nutrition is to be employed rather than breast-milk of a doubtful quality. That rickets is produced by too protracted nursing has not been proven (Siegert), but owing to the deterioration of the milk after prolonged lactation it is not advisable. *Over-feeding* should of course be avoided in rachitic children as well as in healthy ones, but that gastric dilatation due to it (Comby) or chronic dyspepsia are causes of rachitis is unfounded. In *artificial nutrition* the same standard must be maintained as in breast-feeding, namely, that it corresponds with the generally established principles of nourishment of to-day. Whether the milk be sterilized or Pasteurized, or whether any of the many milk preparations (Backhaus, Biedert, Gärtner, etc.) is given is immaterial provided the necessary care is employed. The common fear of starches (infant foods) and the early employment of soups, scraped meat and eggs is no more justified in rickets than at other times. Without any theoretic reservations, the employment of infant foods in the first months of life, and of soup and meat in healthy children younger than six or eight months or even a year is unwarranted. Briefly, in rickets the nutrition is to be the same as an experienced physician would prescribe for any child.

Of greater importance in the treatment of rickets are *air and light*.<sup>1</sup> Here if the circumstances permit, the physician who discovers suspicious symptoms in the new-born may accomplish much by ordering a large, airy room and an out-door life. Particularly in children who are born in the autumn and winter a certain amount of energy is necessary on the part of the physician to make it clear to the parents that the benefit of fresh air to the health of the child is far greater than the danger of "taking cold." Fresh air does not mean that the child is to be tightly wrapped and held so closely that it must inhale the emanations from the nurse. In bad weather the child should be dressed warmly and kept for some time at an open window in a heated room. If the room is large and bright it should not be the resort for the entire day of the

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<sup>1</sup> Seclusion from fresh air is a more important cause of rachitis than poor feeding. Statistics from northern cities, in which well-to-do families house their babies 8 months every year on account of the long duration of their winters, appear to prove it. On the contrary, it struck me while amongst the poor population of Naples, who gave their children scanty and bad food, but fresh air twenty-four hours every day, that rachitis was not perceptible. On the other hand, I am not prepared to say that "protracted nursing is no cause" of rachitis. After the sixth month of nursing when the stored-up iron of the liver in the baby has been used up, the milk of the mother begins to lack some of its needed constituents. That is why I teach the addition of a small amount of cereals and of an animal broth at that age. That is why I am afraid there is a minute degree of levity in the author's remark that "soup and meat in healthy children younger than 6 or 8 months or even a year are unwarranted." I suggest that there is a difference between soup and meat, and another difference between "younger than 6 or 8 months or even a year."—EDITOR.

family and of the seamstress, who vitiate the air. In any case, it must not be forgotten that renewal of air is more necessary for nurslings than for children who are able to walk and thus can vary their surroundings.

In well-to-do families this does not exhaust the care of children with conspicuous rachitic symptoms. On the contrary, a *residence in the country* is to be chosen. The sea-coast or mountains of not too great altitude, and perhaps treatment with brine-baths, are advisable.

For *sea-baths* the Austrian coast resorts on the Adriatic are regarded as beneficial, and from May to October a constantly increasing number of rachitic children are found there. The more important of these resorts are Grado, Abbazia, Lovrana, Sistiana, and Porterosa (near Triest); Lido, near Venice, is also popular. In Germany the Baltic Sea is desirable. Among the many places may be mentioned Heringsdorf, Misdroy, Kolberg, and Ahlbeck. The North Sea is less valuable for rachitics of three or four years of age.

When a residence on the sea-coast is impracticable, regions with a *sub-Alpine climate* during the summer are no less beneficial, especially if there is an opportunity for natural brine-baths. In the Sanatorium for Rachitics near Zürich (750 meters above the sea level), to which children as young as five months are admitted, the results have been very favorable. In a few severe cases under one year, however, there were serious accidents of acclimatization (Hürlimann, quoted by Feer), so that it is better not to send infants and very delicate children at once to a high altitude, but to gradually accustom them to the change. In general a height of about 800 meters is preferable, but strong rachitics over a year old flourish in sunny regions at an altitude of 1800 meters (Feer).

If it is desired to combine *mountain air with brine baths* many well-known regions are at our disposal, such as Aussee, Ischl, Ebensee, Hall (in Austria), Reichenhall, Berchtesgaden, Kreuznach, as well as Kösen, Rothenfelde, Suderode, Elmen, Salzungen, Wittekind and Sulza in Germany (the last quoted from H. Neumann). In some of these resorts there are sanatoria for rachitic children. However, undue importance must not be attached to natural brine-baths; in dry, sunny climates of moderate altitude, artificial salt-baths will be sufficient.

Among the poor, during the summer months, much stress should be laid on the necessity of an out-door life, although naturally in large cities the dust-laden air upon hot days can scarcely be regarded as a curative factor. The physician must make the parents understand that the expenditure of money for a country home for their rachitic children until they are three years of age is often of much greater advantage than later, when they are going to school.

That much may be done for the poor in free institutions which admit rachitic patients for the day or permanently has been shown by the results in the well-known Italian institutions for rachitics (Milan, Turin, Palermo, Genoa, etc.), in seaside hospitals, as well as in the above-mentioned institution in Zürich. In this direction much more might be accomplished, for of late the



hospitals for scrofulous and rachitic children are largely filled with those suffering from the former affection, which precludes the admission of rachitics. Also the age limit of four years of some institutions excludes a number of florid cases. The lack of a sufficient summer vacation is certainly a factor in the late forms of rachitis which are particularly manifest among the poor population.

Among the valuable antirachitic remedies are *salt-baths*, the action of which has been proven practically rather than theoretically understood.

They may be given to children in the first months of life. The bath of an infant—30 to 40 liters—contains about  $\frac{1}{4}$  to  $\frac{1}{2}$  kilo of salt; in baths of 100 to 200 liters, 1 to 2 kilos of salt. Rock salt, "*Halleiner Mutterlaugensalz*," Kreuznacher salt, Stassfurter salt, etc., may be employed; table salt is too expensive for such use. Some manufacturers furnish salt in compressed form for the bath. Coarse rock salt is often contaminated with stony elements; therefore it is well to dissolve the quantity intended for the bath in hot water so that the sediment can be retained. Where natural brine-baths are available they are to be preferred.<sup>1</sup> The stay in the rock-salt bath should be ten to fifteen minutes, the temperature from 25° to 27° C. (77° to 80.6° F.). For older children a sponging with cool water is advisable immediately after the bath. More than three baths a week do not appear to be indicated, and it is well to follow Vierordt's rule, not to continue them for months uninterruptedly, but to give them in courses of six to eight weeks.

At the seashore not only are sea-baths of advantage, usually with warm seawater, but the pure salt air is necessary.

*Aromatic additions to the bath* are alternated with the salt baths (herb and other medicated baths, Feldau's "Kiefermoor"). In cases of rickets dominated by anemia, iron-baths (one small Glob. martialis to a bath) or iron bog baths (Franzenbader Moorsalz,  $\frac{1}{8}$ — $\frac{1}{4}$  kilo. per bath) may be employed. Finally, *sand-baths are very beneficial*, i. e., placing the child in dry sand which has been well warmed by the sun.

This dietetic treatment in the milder cases is often sufficient.

In the *drug treatment* of rickets *phosphorus and cod-liver oil* are the most influential. These were introduced in the treatment of this affection by Kassowitz in 1883. The theoretic foundation for their employment was furnished by the investigations of Wegner, who noted a more rapid consolidation of bone in growing animals after the administration of small doses of phosphorus. Upon the basis of animal experiments Kassowitz has reached the conclusion that phosphorus produces a local inhibition upon vessel formation at the osteochondral boundary and thus acts directly against the inflammation which he supposes to exist.

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<sup>1</sup>The quantity depends upon the concentration of the brine in various districts, as well as upon the size of the bath-tub. In Ischl 4 to 5 liters are calculated for a baby's bath, 15 to 20 liters for the bath of an adult, increasing by one liter per week for infants and 5 liters for adults.

The prescription which Kassowitz prefers is as follows:

R.

Phosphori .....	0.01
Ol. jec. aselli .....	100.0
Ad. vitr. flav.	

Sig. One teaspoonful daily.

The following prescriptions for phosphorus for the most part originate from Kassowitz and are especially indicated when there is great repugnance for cod liver oil:

R.

Phosphori .....	0.01
Liparin .....	100.0

(Liparin, which is quite expensive, may be substituted for cod liver oil in all of the following prescriptions.)

R.

Phosphori .....	0.01
Ol. jec. aselli .....	100.0
Saccharini .....	0.05
Ol. citri .....	gtts. duas

R.

Phosphori .....	0.01
Ol. jec. aselli .....	30.0
Pulv. gumm. Arab.,	
Sacchari āā.....	15.0
Aq. dest. ....	40.0

Or, without cod liver oil.

R.

Phosphori .....	0.01
Ol. amygd. dule. ....	10.0
Pulv. gumm. Arab.,	
Syr. simpl. āā.....	15.0
Aq. dest. ....	60.0

A preparation which is well borne but will not keep.

In the form of drops:

R.

Phosphori .....	0.01
Ol. amygd. dule. ....	10.0
Ol. cort. aur. ....	gtts. iv-v

Three drops in milk three times daily.

For older children chocolate coated tablets of phosphorus may be given:

R.

Pastilli phosphori,

Chocol. obduc. .... .ã 0.0005

One tablet daily.

In all of these mixtures the daily dose of phosphorus is one-half milligram; more is not desirable. A 100-gram bottle will last about three weeks. The administration of the remedy is to continue for several months. During the hot season it is well to stop the medicine—except in threatening cases (laryngospasm)—and the mixture should be kept in a cool place.

An absolutely necessary method of dispensing is Kassowitz's proposition, to keep an oily original solution of 0.2 phosphorus to 100 grams of almond oil, the mixture being dispensed in 5 gram vials.

So much for the administration of the remedy. *But what about the success of phosphorus and cod liver oil?* We now enter into one of the most debatable realms of modern therapy which develops a rather unusual circumstance, i. e., that, on the one side, a remedy is regarded as an absolutely reliable specific, and, on the other, as entirely valueless.

Kassowitz champions his remedy with great energy and does not tire of combating the publications opposed to his view. His absolute confidence in the drug is based upon his animal experiments, his rich clinical experience, the high praise of other prominent pediatricists, and upon the enormous popularity which the remedy possesses among the poor population of Vienna. To anyone acquainted with the conditions in Vienna it is remarkable how often mothers bring their children to Kassowitz's Institute with a particular request for a prescription for cod liver oil, and that regular attendants at other children's clinics prefer to pay for the medicine at the pharmacy attached to Kassowitz's Institute rather than obtain it free of cost from their own dispensary. It is also extraordinary how many prominent specialists in children's diseases favor and even praise the actual success of the remedy without accepting Kassowitz's theoretic explanation of its effect.

Opposed to them are quite a number of physicians who are either skeptical or deny the value of the remedy. Among them are Henoeh, Heubner, Baginsky, Monti, H. Neumann, Comby—authorities to whom we cannot attribute mere prejudice or deficient faculty of observation or limited clinical material.

The arguments which refute the *action of phosphorus and cod liver oil* are of different value.

Least important, probably, is the objection that phosphorus is very soon volatilized from the cod liver oil mixture so that the child really receives a smaller amount than has been prescribed. Careful investigation of drugs from reliable pharmacies have constantly shown a positive reaction of phosphorus even when the bottle was uncorked for some time. However, if this were not the case no conclusion should be drawn against its effect, for to obtain success from the treatment, which is prolonged for several months, it is irrelevant



whether a smaller amount of phosphorus is sufficient than was intended in the prescription.

Clinical considerations are more difficult to meet. It is mentioned that the rachitic bony process tends to spontaneous cure; therefore, even if there has been improvement in the disease of the skeleton after phosphorus has been administered for weeks or months, the effect of the drug has not been proven. On the other hand, cases treated with phosphorus do not always show improvement. Finally, it is still undecided whether, in the cure of the rachitic bone disease, a primary influence upon the skeleton is exerted or a secondary consequence of improvement in the general condition. Unanimity on these points appears to be impossible, since experience is opposed to experience and views are opposed to views. If, for example, in a rachitic, toothless child, a tooth appears after the administration of phosphorus for eight or ten days, this is a curative effect of the remedy for adherents, while the opponents regard it as an accidental occurrence on account of the brief treatment. If a fontanelle closes after several months' administration of phosphorus some authorities believe this to be the result of continued treatment, others the natural issue of the disease. Mere clinical observation of the skeleton, therefore, furnishes no positive proof of the specific action of phosphorus and the objections of the opponents of phosphorus are hard to refute.

An attempt to meet this question by another was made by Stöltzner, who admits the favorable effect of phosphorus upon the ossification of bone, but, from anatomical investigations in animals, has taken the stand that the remedy brings about an improvement of the osteoporosis which is present to such an extraordinary degree in rickets without influencing the pathognomonic osteoid tissue. This would prove the activity of phosphorus in strengthening the bone, but will not confirm the theoretic considerations which Kassowitz has attached to this process of healing.

A further question concerning the action of phosphorus and cod liver oil is whether *the cod liver oil does not have a greater therapeutic value than phosphorus*. It must not be overlooked that no less an investigator than Trousseau strongly advised cod liver oil as a remedy for rickets, that prior to the introduction of phosphorus in the treatment of this disease the advocates of pure cod liver oil were almost as numerous as those at the present time for phosphorus. Even now the converts of phosphorus adhere to its combination with cod liver oil and some of them believe that phosphorus alone, without cod liver oil, does not completely unfold its activity. A procedure which would perhaps settle this question was proposed by Rey, who tested the influence in the rachitic child of phosphorus alone and of cod liver oil alone, and of their combination, upon the excretion of urate of calcium. It was shown that administration for several days of the combined remedies always increased the amount of calcium urate, which did not occur when the mixture was withdrawn. Phosphorus without cod liver oil had no effect. Cod liver oil alone caused a slight increase of calcium urate. These investigations, which would indicate a special action of a combination of phosphorus and cod liver oil, are unique and as yet

inconclusive. Vierordt, who inspired Rey's investigation, warns against too positive deductions.

In this wordy combat regarding the value of phosphorus as an antirachitic remedy two things are quite certain: *the often surprising success of the remedy in spasm of the glottis and the praise of the parents of rachitic children for the medicine.* When laryngospasm which has lasted for weeks disappears after two or three teaspoonfuls of a phosphorus-cod liver oil mixture, and does not recur, it cannot be regarded as other than a therapeutic success, which is not lessened by its occasional failure to act—particularly in older children with expiratory apnea. It has already been remarked that this result is apparently no proof of the antirachitic value of phosphorus, but follows a direct influence upon the nervous system. Somewhat less conspicuous is the effect of phosphorus and cod liver oil upon tetany and upon the convulsions, and still less upon spasmus nutans.

The popularity and favor which phosphorus and cod liver oil enjoy among the poor of Vienna is certainly a factor not to be undervalued in judging their effectiveness. It is questionable whether a conclusion can be drawn from it regarding the improvement of the rachitic process, but we hear statements of a brighter condition, better sleep, greater activity, and this sometimes eight to fourteen days after the institution of the phosphorus-cod liver oil treatment. It appears here also that the anodyne, sedative effect of the remedy is not of subordinate importance, and, this quite apart from all theoretic considerations, is a chief factor in the popularity of the remedy.

From these considerations it will be seen that the opinions regarding phosphorus as a specific for rickets are divided. This, however, has not prevented its successful introduction throughout the world, and there is scarcely a children's hospital wherein phosphorus and cod liver oil are not prescribed by physicians and accepted with gratitude by the parents of the patient. There is probably no danger in the remedy and the recently reported cases of poisoning from therapeutic doses of phosphorus are too few to cause apprehension from its administration, especially when we consider the widespread use of phosphorus and cod liver oil. It cannot be denied, however, that this medicament frequently has a deleterious effect upon appetite and digestion—particularly in children over one year of age; therefore, contrary to many opinions, the remedy should not be employed when there are disturbances of the digestive tract. Other remedies recommended for rickets are of secondary popularity.

*Cod liver oil* alone, as already mentioned, is often prescribed, and formerly was much employed. If it cannot be taken in pure form the following emulsion may be given (Frühwald):

R.

Ol. jec. aselli .....10.0-20.0

Mucil. gumm. Arab.,

Aq. dest. āā. q. s. ut fiat emulsio ...90.0

Syr. cort. aur. ....10.0

One teaspoonful three times daily.

There are also quite a number of preparations of cod liver oil which contain this ingredient alone (Helfenberg's sparkling cod liver oil, Standtke's palatable cod liver oil, Scott's and Mellin's emulsions, Natterer's cod liver oil tablets), or combined with other drugs (Lahusen's iodid of iron and cod liver oil, Sauter's lecithin-cod liver oil emulsion, combinations with malt—Löflund, Liebe, Hauser & Sobotka—etc.).

If a rachitic child is to be given cod liver oil, the most rational form is in combination with phosphorus.

*Calcium preparations* have lost all theoretic value and appear to be superfluous in the treatment of the English disease. The calcium-iron syrups which are still used in rachitis owe their introduction most probably to the seductive word calcium.

The importance of *iron* in rickets is quite different. Although phosphorus and cod liver oil are usually employed for the pallor, in many children this remedy is not well borne and iron is substituted. Iron preparations may also be alternated with phosphorus and cod liver oil (particularly in summer). Finally, by those who doubt the efficacy of phosphorus in the treatment of rickets, iron is employed and its action is praised. The most popular form is *iodid of iron* in the following combinations:

R.

Syr. ferri iodid  
Syr. simpl. ....ãã 10.0

Ten drops several times a day for children of four to seven months; 15 drops for children of eight to twelve months; 20 drops for children two years old.

R.

Syr. ferri iodid ..... 5.0  
Syr. simpl. ....20.0  
Aq. dest. ....80.0

Two to four teaspoonfuls daily or malt extract with iodid of iron,  $\frac{1}{2}$  to 1 teaspoonful twice daily.

Other preparations of iron are:

R.

Tinct. ferri chlorid .....10.0

Eight to 10 drops three times daily (Hench).

R.

Ferri lact. reduc. ....0.03-0.05-0.1  
Sacch. lact. .... 0.30

One powder three times a day or *ferratin*, *fersan*, *alboferrin*, *nematogen* (Hommel), *hematicum* (Glausch), *iodferratose*, *Gude's peptomangan*, and many others.



Lately *sanatogen* (casein with glycero-phosphate of sodium) has been given as a food to rachitic children.

Owing to the theoretic considerations which attribute rickets to the absence of some internal secretion, a number of *organo-therapeutic tests* have been made. Preparations of *thyroid gland* (Knöpfelmacher, Heubner), of *thymus gland* (v. Mettenheimer, Mendel), and of the adrenals ("*rachitol*," Stöltzner) have been employed without convincing results.

Certain of the individual *symptoms* of rickets require special therapeutic consideration.

For example, on account of the ready flexibility of the skeleton, rachitic children should in general be kept at rest as much as possible. Too early standing and walking should be avoided. Restless infants with craniotabes should lie on hollow cotton pads; the bed of a child with *kyphosis* must be hard and without a pillow, or Rauchfuss's apparatus may be used; those with beginning scoliosis should be carried on each arm of the nurse alternately; finally, if curvature of the legs develops, proper orthopedic apparatus must be employed.

A very useful appliance is *Epstein's swinging chair* for rachitic children (manufactured by Thonet), in which the patient—properly supported by braces at the back, on the sides, and at the feet—is so placed that he sits with his face toward the support and must balance the body.

The treatment of marked rachitic curvatures of older children belongs to the realm of orthopedic surgery. Surgical intervention for knock-knees or bow-legs should not be considered hastily, for spontaneous cure may occur as late as the sixth year.

Frequently advised, but seldom utilized, is the inhalation of condensed air for the improvement of respiration and to compensate for the deformity of the thorax.

The *accompanying symptoms and the sequels* of rickets require suitable treatment. This applies especially to diseases of the respiratory and digestive organs. In acute respiratory disturbances phosphorus and cod liver oil are indicated. That this form of medication is undoubtedly necessary for the nervous phenomena has already been mentioned. Only when phosphorus is not effective are other nervines, such as bromin, to be employed.

# INFANTILE SCROFULOSIS AND TUBERCULOSIS

By O. SOLTSMANN, LEIPSIK

## RELATION OF SCROFULOSIS TO TUBERCULOSIS

Scrofulosis and tuberculosis, two inseparables! Who can mention one without acknowledging the other? The first is the precursor of the second—plows and opens the soil for it; the relation is that of the seed and fruit to the fostering soil, of the soil from which the plant grows to the plant itself, it is a part of the plant, the essence of the root, its extract—and all the other synonyms which denote their connection and intimate relation, and which have been mentioned to force the general recognition of the *monistic theory* in opposition to the dualistic view.

Nevertheless, with our deeper insight, or perhaps in spite of our deeper insight, of the development and nature of tuberculosis, the task of the Unitarian enthusiasts is not easy.

Proceeding from the caseous products of scrofulosis and tuberculosis, Laennec saw in the former only a localized glandular tuberculosis. Virchow took a decided stand against such an opinion. He distinguished the actual tubercle from the irritative, inflammatory, hyperplastic processes of the tissue masses which originated in caseation, particularly of the lymph glands, of which scrofulosis is the chief representative. The decisive factor in tuberculosis is the organized cellular, non-vascular, heteroplasmic new formation. It is true, even this undergoes a regressive, fatty, caseous metamorphosis (with a loss of its activity) by cell proliferation and vascular compression, but it is not limited to a definite pathologic tissue, on the contrary, being present in very heterogeneous products. The tubercle alone decides whether or not the process be tuberculosis.

This at first furnished a new foundation for the *dualistic theory*. When the miliary tubercle, with its epithelial cell accumulation and central giant cells, was also found elsewhere, and its inherent specific nature could no longer be maintained, we gradually came to realize that the anatomical definition alone did not suffice for tuberculosis. The morphologic equilibrium, on the one hand, does not prove the essential unity, and, on the other hand, the etiologic unity does not prove a similar histology. Cohnheim was the first to regard caseation and the tubercle as non-specific for tuberculosis except that both were the product of the tuberculous "virus," that is, that their experimental inoculation produced tuberculosis! This placed the question of tuber-

culosis upon an entirely new footing. Instead of the humoral and cellular conception of the nature of tuberculosis or of scrofulosis, the etiologic factor—infection—was substituted. It is true, this then was not new, for there was no doubt of the contagiousness of tuberculosis and scrofulosis; and the inoculation of tuberculosis from tuberculous products, i. e., caseous glandular material, had long been known. The pioneer experimental investigations of Villemin—Buhl's law of tuberculosis as a disease of absorption—were sufficient to fasten the conviction of tuberculous infection, but only after the labors of Tappeiner, Schwenninger, Lippe, Bollinger and Cohnheim did tuberculosis as an infectious disease receive "official sanction."

However, in all of these investigations, where was the confident criterion that the material employed in inoculation was actually and only the carrier of the tuberculous virus, and of what nature was this virus? With the discovery of the tubercle bacillus by R. Koch (Baumgarten), the law of the infectiousness of tuberculosis reached its final acceptance. R. Koch taught how to differentiate the tubercle bacillus from all other schizomycetes. He further taught how to produce pure cultures outside the body, further, the transmission into various animals of bacilli of several generations which showed undiminished virulence, and proved that all pathogenic products developed by the tubercle bacillus were actual tuberculosis. He again restored the tuberculous nodule to its proper position for the anatomist, as Cohnheim had hoped. For Koch had recognized that the tuberculous nodule was a true infectious tumor, that the miliary nodule which produced proliferated inflammation of the fixed tissue cells, terminating in caseous necrosis, was the product of the tissue change brought about by the parasite, and that it was even the first effect of the action and proliferation of the bacilli after their local accumulation. Koch was succeeded by Demme, Krause, and many others, who noted that tubercle bacilli were present in a number of affections of the skin, glands, bones, and joints, which were formerly regarded as specifically scrofulous. Then the uncertain foundation upon which the dualists stood appeared to collapse and leave no question of the *identity of scrofulosis and tuberculosis*.

Nevertheless, two elements were overlooked: First, in many such cases it was impossible to exclude, in fact it was very reasonable to assume, that the tubercle bacillus was now and then only an accidental host in the scrofulous focus; that accordingly, when it accumulated in this region it apparently played but a secondary rôle, in that the preceding scrofulous infection had furnished it a point of entrance and of settlement; second, Koch himself had frequently failed to demonstrate, in the softened scrofulous glands, the epithelioid cellular nodules and giant cells, tissue necrosis and bacilli. Further, tubercle bacilli could not be found in the numerous peripheral, primary, circumscribed and diffuse scrofulous affections of the skin, mucous membranes and the glands at any time during their existence. I refer to conjunctivitis, rhinitis, to ozena and otitis, to the various forms of scrofulous eczema, to the common tenacious and deep cutaneous abscesses and serpiginous ulcers, and to the fungoid, osseous, and joint affections which had always been regarded as tuberculous.



That the tubercle bacillus had been present in all of these affections, and had disappeared, is quite untenable, at least for the incipient and progressive processes, and the objection cannot be opposed that even in those scrofulous affections wherein tubercle bacilli were found the absolute proof "that these were originally and exclusively of a bacillary-tuberculous nature" is still lacking. Biedert adds that such a conception is impossible.

If, therefore, it is necessary to find in the tubercle bacillus alone, or also in the proliferating nodules, the criterion—whether or not in a given case tuberculosis is present—the tuberculous nature of scrofulosis is not yet proven; then, in this sense at least, both are not identical.

In such a decision another factor must be invoked—*heredity*! This to a certain extent represents the highest potency of infection. The belief in the heredity of scrofulosis and tuberculosis was regarded from ancient times as an axiom, based mainly upon the great number of scrofulous and tuberculous children descended from parents affected by these diseases. The chief exponent of the absolute heredity of scrofulosis was Lugol; of tuberculosis, no less a one than Baumgarten. Even Cohnheim was imbued with the magic spell of the heredity of tuberculosis as an event which had been proven by thousands of experiences. Liebermeister, Nocard, Knopf, Haupt and others maintained the same view, and Haupt reckoned among 1,753 phthisical patients 1,197 who were hereditarily predisposed. Tuberculosis was therefore an eminently hereditary disease.

It is a remarkable fact that the belief in the heredity of scrofulosis became more prominent equally as the belief in its contagiousness declined. Quite the opposite was the result in tuberculosis. The better we learned to realize the contagiousness of the latter affection by the enormous increase of cases, the more did the belief in its heredity lose ground, both, I believe, with a certain degree of justice, as the consequence of a more thorough knowledge of the tubercle bacillus and its biologic properties.

The proof of the heredity of tuberculosis presupposed that fetal bacilli and nodules could be demonstrated, or at least that fetal tuberculosis could be produced experimentally. Both conditions have been fulfilled. But this is true only of the placental, not of germinative heredity. Johne was the first to demonstrate tuberculous foci, with epithelioid and giant cells, and tubercle bacilli in a fetal calf, these being principally localized in the liver and portal glands and proving unquestionably the placental source of infection. Similar cases were reported latter by Malvoz, Brouvier, Lungwitz, Band and others. Gärtner and de Renzi, in their well-known experimental animal researches, positively demonstrated the transmission of tubercle bacilli from the mother to the offspring, and Gärtner, in his experiments which were capable of withstanding the sharpest criticism, came to the conclusion that the tubercle bacilli frequently passed to the young from the mother, not from the father—but from the latter by the sperma to the mother (genital tuberculosis). In the human being, thus far, only the placental, not the germinative (spermatogenous, ovogenous), heredity of tuberculosis has been observed and proven, and

must be regarded as a rare occurrence, notwithstanding all the statistics at hand. Scarcely two dozen cases can be found in the entire literature which can be designated as positive (Birch-Hirschfeld, Schmorl, Kockel, Lehmann, Baumgarten, Roloff and others). Lehmann at this time reported the first case of development of placental tuberculosis with transmission from the pars materna to the chorion villi, while Schmorl and Kockel were able to follow the advance of placental tuberculosis from the intervillous spaces to the chorion villi and thereby demonstrate the course into the fetal circulation. We know, however, that in previously reported cases of congenital tuberculosis the placenta itself was diseased. This is important, for the placenta physiologically forms a filter which retains corpuscular elements and permits their passage only when this organ is diseased, eroded, its epithelium injured, etc. On the other hand, the passage is free for soluble substances, even in an intact placenta. This is all the more noteworthy since it is known that we dare not assume a specific infection simply by the demonstration of specific pathogenic agents, that the products of metabolism (toxins) of pathogenic germs of disease are capable of producing permanent infection (intoxication) by their chemotactic action.

For example, we know that in tetanus and diphtheria some pathologic picture or intoxication may be produced by the cultures of the specific and entirely destroyed attenuated bacteria, or even by preparations which have been made from them by means of chemistry, such as the bacteria themselves produce in animal and man; and it is certainly correct to assume analogous conditions for the chronic infectious diseases. A certain number of what we to-day regard as "general nutritive disturbances, constitutional anomalies, diseases of metabolism and the blood" may be referred perhaps to a development of this kind, as *hereditary habitual infectious diseases*, from the fact that, in the soluble products of metabolism, pathogenic germs (toxins) have passed through the placenta and have been transmitted from the mother to the fetus. This perhaps is the condition in tuberculosis and would therefore give us some conclusions regarding the relation of tuberculosis to scrofulosis, of the mother—to the daughter—disease, and of the nature of the latter. If, as we have seen, a transmission of bacillary tuberculosis is so exceedingly rare, is it necessary to exclude a non-bacillary form? By analogy, the opposite may be assumed from what has been stated, and *scrofulosis perhaps represents this non-bacillary hereditary toxic tuberculosis due to pathologic chemotaxis*, which explains the descent of scrofulous children from phthisical parents. This would also make clear the great similarity of the symptoms, the wide variation of the course and termination, and why, in the majority of cases, scrofulosis terminates in recovery, but under some circumstances runs a fulminant course with sudden aggravation due to secondary bacillary infection, and shows an unfortunate outcome. The conception of scrofulosis as a *non-bacillary hereditary tuberculosis* gains in force from our knowledge of the action of the tubercle toxins. This culminates in signs of inflammatory reaction and also in caseation.

The experiments of Wyssokowitsch, Prudden, Vissmann, Strauss, Masur, Stroebe and others have shown that after the inoculation of attenuated tubercle bacilli a variety of miliary tuberculosis may develop which differs from true miliary tuberculosis only "by the deficient infectiousness." Caseous nodules also develop as in the local proliferation of tubercle bacilli. I must not fail to mention that the development of caseous pneumonia is referred to the action of soluble products (tuberculin), to which, to a certain extent, even a protective action against bacillary infection is imputed. For the correctness of the foregoing views regarding the nature of scrofulosis and its relation to tuberculosis it is quite possible to obtain further support by experiments upon suitable animals.

In this form of infection, what is known in scrofulosis as a hereditary predisposition to tuberculosis I would designate the *hereditary diathesis*, of which I am concerned only with the time of appearance and the nature of the manifest specific sequelæ; how far in this process of transmission the fruit—the fetal organism—has at disposal protective substances, "bactericidal material (alexins), in contrast to the attacking substances (lysins) arising from virulent bacteria of the maternal organism."

We may even admit, to a certain degree, a predisposition to tuberculosis in scrofula, in so far as it produces a favorable soil for a later accumulation, in contrast to the parasital heredity, which is itself of a bacillary nature (Lubarsch). What we term "habit" in scrofulosis is therefore the expression for the permanent condition, for the "*habitual infectious disease*" itself, such as is shown in its external and internal phenomena, according to age, structure of the body, manner of life, etc. I do not agree with Cornet, who, in his admirable work upon scrofulosis—wherein he cannot deny a certain uniformity—attempts to differentiate two forms of scrofulosis: the tuberculous, due to the tubercle bacillus, and the non-tuberculous, the development of which is caused by other, especially pyogenic, bacteria, and to these adds a mixed form. Scrofulosis in itself has as little to do with streptococci and staphylococci as tuberculosis, diphtheria, and other specific infectious diseases, although there is a plentiful opportunity for the absorption of inflammatory organisms through the skin, mucous membrane and lymph tract, by way of the numerous local eruptions at the various points of entrance. That these by no means have a specific character corresponding to the primary disease, and that, on the other hand, in their symbiosis and synergesis with the specific pathogenic agents, they present such a destructive tendency, such a malignant life-threatening progression, is sufficiently well known as in diphtheria for the acute, and in tuberculosis for the chronic, infectious diseases.

Such a secondary rôle is enacted in the case of scrofulosis, not only by pyogenic organisms, but by the tubercle bacilli themselves. Cornet maintains that in scrofula the deleterious agent constantly finds its entrance from the periphery, and therefore he holds that the nature of the scrofulous predisposition should be referred to the periphery of the body. He does not believe in a predisposition which dominates the entire organism of the individual, but attrib-



utes the appearance of scrofulosis in childhood mainly to the local anatomical relation of the protective covers with increased permeability of the skin, mucous membrane and lymph tract,—a condition which he designates “*embryonalism*.” Hueter considers the great width of the canals, through which inflammatory agents may readily reach the lymph tracts and thence be transported into the lymph glands, as the cause of scrofula; he regards lymphadenitis as the consequence, not as the first expression, of scrofula. The pasty, spongy appearance of scrofulous children is simply the result of an over-filling of the connective tissue and of the canals with nutritive juices. Ponfick agrees partly with Cornet, partly with Hueter. He desires to maintain the conception of scrofula independently of that of tuberculosis. He also assumes, upon a constitutional basis, a pyogenic and tuberculous, and a reciprocal combination of both forms, in scrofulosis, wherein he ascribes the more ready invasion of bacterial germs to the greater richness in juices, the greater distensibility of the lymph spaces, the more active chemotaxis of the leukocytes, and the greater faculty of proliferation of the infantile tissue, causing on the one hand the peculiar swelling, and, on the other, the tendency to exudative processes and proliferative reaction, which develop the peculiar picture of scrofulosis. In how far these views coincide with mine is obvious.

## SCROFULOSIS

Nevertheless, under the influence of numerous factors, which were formerly designated as the cause of scrofula, we may particularly understand that, according to the concentration and action of the chemical products of metabolism of the tubercle bacilli in the blood and lymph systems, the condition appears that is commonly recognized in the picture of scrofulosis as the *erethistic and torpid habitus*, by which we mean the habitus as an expression of the disease itself.

**Torpid scrofulosis** is characterized by the coarse, thick and broad lines of the face, the swollen nose, the turned-up, thickened lips, the visible hyperplastic lymph glands upon the throat and nape of the neck, the distended obese abdomen, the flabby flesh, the spongy, edematous tissue. In **erethistic scrofulosis** we note the soft, silky hair, the languid eyes, the blue sclera, the reddened cheeks, the bluish-white glistening teeth, the delicate skin permeated by a blue network of veins, the thin, delicate muscles and bones, the graceful contour of the body, and the slight amount of fat. The torpid form presents the characteristics of a *retarded* metabolism, with a sluggish, phlegmatic temperament, the erethistic of a greatly *increased* metabolism, with nervous excitement.

And now the *polymorphic varying nature of scrofulosis*? How can we describe the **local phenomena**, in their common fundamental relation, better and more aptly than Virchow? They culminate in the vulnerability of the tissue with inflammatory changes of the skin, mucous membrane, glands, bones, and joints; they reach their acme in the tenaciousness of their existence, in their exacerbations and remissions, in their coming and going, in constant

relapses without cause, in the multiplicity and combination of the local foci of the skin, mucous membranes, glands, etc. This is characteristic of the eczema, which appears usually over the face, on the cheeks, chin, mouth, nose, eyes, ears, more rarely over the hairy scalp and the trunk; sometimes papular, sometimes vesiculo-pustular, at other times crustaceous and squamous, according to its location, and characterized by its tenacity. At the openings of the organs of sense the eczema may be complicated by like affections of the mucous membrane which sometimes extend to the eczema, or, inversely, the corrosive secretion of rhinitis and coryza may reach these areas and give rise to crustaceous fissures and swelling of the upper lip. In furunculosis of the auditory passage the eczema may produce a catarrhal or a seropurulent otitis which finds its way over the structures of the ear and causes a moist eczema rubrum upon the aural cartilage with infiltration, thickening and fissures of the red, glistening skin. Further, in conjunctivitis the eczematous secretion which oozes from the glued eyelids may produce blepharadenitis, with photophobia and blepharospasm, and cause severe pain and bleeding fissures at the palpebral angles and over the cilia and eyebrows, or there may be a moist eczema of the cheeks or pustulation around the hair follicles.

In themselves these **affections of the skin and mucous membrane** are not all specific, but their tenacity, which distinguishes them from similar affections which are opposed to remedial agents, their partial disappearance, or absence for a brief time, their unexpected return without cause in the same or an adjacent area, with a renewed tendency to attack other parts, as mentioned above, the implication of the neighboring lymph glands with hypoplastic swelling, are all characteristics which bestow to the affection its scrofulous physiognomy. But scrofula of the skin does not take the form of eczema alone. Acne, furunculosis, lichen, even lupus and scrofuloderma, which, in part at least, are forms of primary cutaneous tuberculosis; also the subcutaneous cellular tissue abscesses with their relapses, fistulous ruptures, excavated and tenacious ulcerations, are peculiar to scrofulosis and cannot be designated as tuberculous. Although Demme frequently found tubercle bacilli in the scrofulous eczema of children we would not for this reason presuppose the eczema to be tuberculous. Only when the bacilli accumulate and proliferate rapidly, and develop their biological properties, are we justified in considering tuberculosis, and then it is a secondary cutaneous tuberculosis planted upon an eczema.

*Affections of the mucous membrane of the eyes, the ears, and the nose*, the common tenacious forms of *angina pultacea* and *angina pustacea* with their pasty plugs and glassy purulent production of mucus from grooved *hypertrophic tonsils*, the *pharyngitis granulosa* with adenoid proliferation of the *nasopharyngeal space* and of the hyperplastic pharyngeal tonsil, the constantly relapsing *catarrh of the bronchi* and of the *gastro-intestinal mucosa*, with dyspeptic disturbances and habitual constipation, are *manifest stages of florid scrofula* and are characterized by their great tenacity, just as are the non-gonorrheal sero-purulent secretions of the vagina with inflammatory infiltration of the labia, especially in young persons.

**Affection of the glands.**—The entire picture of scrofula is, however, dominated by the *glandular affection* which is often the first sign of the disease and not infrequently exists alone, without secondary symptoms. Here the acute inflammatory swellings known as “the concentual bubo,” and regarded as a *secondary disease*, particularly in scrofula, are of less consideration in connection with hyperplastic inflammatory diseases of the skin and mucous membrane than the *chronic hyperplastic swellings, independent of any local process of the skin and mucous membranes*, which appear very much later and arise insidiously, without fever and without pain. They are a *main characteristic of the picture of scrofula*. Here, upon the basis of the above diathesis, we are not dealing with inflammatory swellings occurring inwardly from without, but with those appearing externally from within. These in the true sense of the word are “poison glands,” true hyperplastic lymphomata with interstitial connective tissue proliferation.

We find these *peripheral glands* enlarged—in contrast to the *visceral enlargements in tuberculosis*—in the nape of the neck, the jaw, the throat, more rarely in the axilla and inguinal region. They are often the size of a pea or a bean, and may attain the size of a pigeon’s egg or a walnut, constituting tumors and glandular conglomerations over which at first the skin is normal and movable; later, however, the cutaneous covering is reddened and adherent to the gland. These glandular tumors fluctuate in size, may retrogress and for a long time remain unchanged, without resorption, but they invariably recur and slowly and insidiously undergo change, showing caseation, softening and pus formation, and finally *rupture externally* with the discharge of milky purulent material mixed with caseous flocculi. Occasionally there are sinuous fistulæ due to ulceration, which heal, leaving disfiguring cicatrices with striated sclerotic lines in the retracted tissue.

Under such circumstances pyogenic and other bacteria readily find a port of entrance, especially if there are local inflammatory eruptions in the adjacent skin and mucous membranes, and it is obvious that softening and supuration of the gland is thereby facilitated. It must be emphasized that these entering bacteria have nothing in common with the specific primary affection, but they may place their definite stamp upon the secondarily infected gland. This is the case with streptococci and staphylococci, and also with tubercle bacilli. When there is an invasion of the tubercle bacilli the scrofulous gland also becomes tuberculous. But neither the anatomico-histologic nor the bacterial findings will justify the assumption that all scrofulous glands are tuberculous.

The **scrofulous osseous and arthritic inflammations** which run a chronic course are now generally regarded as tuberculous. The fact that general miliary tuberculosis or tuberculous meningitis develops, often suddenly or spontaneously, after trauma or following some operation attempted as a therapeutic measure, is certainly convincing. We know, however, of a number of prolonged affections which recover under favorable external conditions; and again, in a number of cases, Biedert, Kanzler, Schlegdendahl, Krause and others



could find neither tubercle bacilli nor tuberculous nodules in the osseous foci and fungoid joint masses. Moreover, we cannot state absolutely that the pathologic foci in the bone, bone-marrow and joint were not originally tuberculous, and were only secondarily infected by the bacilli. Henoch expresses the condition as follows: "The enormous growth of bone in infancy, especially at the epiphyses and under the influence of unknown conditions which also give rise to chronic inflammation in other tissues, may all the more readily produce a marked hyperemia and its consequences, without the early existence of tubercle bacilli." He refers to syphilitic affections of the bone, due to an unknown dyscrasic irritation, and believes that this view applies also to the development of bone tuberculosis under traumatic influences.

Decide the question as we will, we cannot alter the *substantive pathologic picture of scrofula*, and at this time there is not a single convincing factor to combine this disease with bacillary tuberculosis. The general appearance, the entire course, and the termination of scrofula favor this view. For no matter how long its duration, often extending over many years, no matter how manifold and varying the symptoms, with periodic improvement and aggravation, notwithstanding the insidious relapses with the same or new local eruptions of the skin, mucous membrane, glands, etc., we observe neither a high-graded anemia nor any particular corporeal weakness; and, under any circumstances, I cannot admit that in scrofulous children a physical debility remains which may be permanent. In fact, if the external surroundings of the patient are favorable, we observe after a period of years, usually about the time of the second dentition (the sixth year) a *tendency to recovery*; and, apart from slight residua (cicatrices and defects) *complete recovery is the rule*, which would be all the more general if secondary and mixed infections were prevented by suitable hygienic measures and prophylaxis. Only rarely do we observe new local eruptions after puberty, although I must agree with Baginsky that now and then relapsing naso-pharyngeal catarrh, hypertrophy of the tonsil, and bronchitis remind us of the scrofula of childhood.

## TUBERCULOSIS

How different the conditions in tuberculosis! Here, in contrast to scrofula, the numerous local phenomena are of minor importance to the constitutional symptoms. This is the more true the younger the child, and in small children, especially nurslings, the disease may run an almost latent course. Nevertheless, tuberculosis demands many sacrifices even in infancy. If we include the period of earliest childhood we may state positively that about 40 per cent. of tuberculous cases show a mortality in nurslings. From the second to the seventh year this mortality is greater, since the local phenomena then became more prominent. At the period of puberty there is a decrease in the death-rate, but in the following years many succumb to the disease, which then runs the course and presents the picture of tuberculosis of adults.

**Tuberculosis of Infancy.**—As already explained, it is difficult to prove the congenital nature of tuberculosis by the frequency of the disease in infancy. We to-day know that the offspring of phthisical parents are not predisposed to tuberculosis; on the contrary, when the child is removed early from the environment of such parents, it is not affected by the disease. This was recognized by Epstein in 1879, and explains the exceedingly small number of cases of tuberculosis in nurslings in the Foundling Asylum in Prague, where the tuberculous mothers leave the institution as soon after labor as possible, their children being given to healthy women. In striking contrast is the report of Froebilius of the frequency of tuberculosis in the Foundling Asylum in St. Petersburg where such measures are not observed—an important indication for prophylaxis. The overwhelming majority of cases of *infantile tuberculosis* are acquired extra-uterine, exogenous from the intimate contact with a phthisical mother or nurse; not, as was formerly assumed, “ingested with the milk,” but inhaled with the respired air. The maternal atmosphere surrounds the nursing like a bacillary spray. Because the child is so intensely and continuously exposed to the danger of infection it is so frequently and so readily attacked, and the source of the disease is more apt to be overlooked than in the older child whose exposure to infection is so much greater. Finally, it is manifest that in this daily increasing danger of tuberculous surroundings, the debilitated, weak offspring of phthisical parents, who perhaps have battled in the womb of the mother for existence, are especially liable to infection.

*Infantile tuberculosis* presents the symptoms of *atrophy* without local phenomena on the part of the respiratory tract. The innutrition, the sudden arrest of development, and subsequent decline of the body weight, with rejection of food, and apathy, the disappearance of the fatty tissue, the sallow, wrinkled, gray skin which has lost its natural turgescence and is covered with flaky desquamation and frequently with intertrigo, furunculosis, and multiple abscesses, the thin, hard strands of muscle standing out like folds, the legs adducted at the knee, edema of the ankle and the dorsum of the foot, the unmotivated dyspeptic symptoms, the mummy-like, shrunken little face, the bones of the skull which overlap in the form of terraces, and the hollowed fontanelles, the sunken eyes, the wide-drawn mouth surrounded by radiating lines—such is the picture of these poor little unfortunates who, without cough and without expectoration, but with a small, frequent pulse and atypical changes in the temperature which is often subnormal, are certain candidates for death. Seldom will we succeed in making a diagnosis from signs on the part of the lungs, or to demonstrate the presence of tubercle bacilli. Epstein and Kaufmann have found tubercle bacilli in mucus which was caught in the opening of a soft catheter inserted in the mouth to the root of the tongue to induce cough.

*Infantile tuberculosis* usually runs a *latent* course because of its *localization in the bronchial glands*—simultaneously a proof of infection by inhalation through the respiratory tract—and the resorption of toxins from these isolated foci occurs so slowly, although steadily, and in such small quantities

that very often instead of a febrile reaction there is an intoxication of the entire organism which results in marasmus and death.

**Tuberculosis of the Bronchial Glands.**—Tuberculosis of the bronchial glands not only occurs in infancy, but, in contrast to tuberculosis of adults, it dominates childhood up to the twelfth year, with a tendency to rapid caseation and propagation; or encapsulation occurs and forms a secret deposit, so that occasionally, when other diseases—as whooping-cough, measles, influenza—quicken these old masses, the material which is capable of propagation is distributed in the organism.

There is unanimity of opinion regarding the enormous frequency of tuberculosis of the bronchial glands in infancy. I calculate its occurrence in my cases which have come to autopsy to be 85 to 90 per cent. In 320 autopsies of tuberculosis in infancy Steiner and Neuretter found the lymph glands affected 299 times, and the bronchial glands 286 times; Rilliet and Barthez 249 times in 312 cases. Henoch, Neumann, Seitz, Baginsky and others report similar findings.

According to Weigert tubercle bacilli choose the same road as coal-dust, which first collects in large masses in the bronchial glands. Cornet is of the opinion that the rapid and early implication of the bronchial glands in infancy is due to the size of the lymph-vessels and tubes, to the keen energy of the lymph stream which is caused by the active metabolism, and to the larger absorption of oxygen and excretion of carbonic acid; thus the bacilli find but little time to accumulate in their pulmonary passage.

Tuberculosis of the bronchial glands is not always latent. At the onset the glands are the size of a lentil or a bean, and when they act as a filter to retain the pathogenic organisms, with their immoderate and steadily increasing focal proliferation—and provided a generalized tuberculosis with a rapidly fatal course does not previously occur—when they are the size of large nuts or even of a fist and consist of glandular conglomerations, when they remain within their greatly thickened capsule and not infrequently form firm adhesions to their surroundings, then it is possible to demonstrate them by physical examination, and when they surround air canals, vessels and nerves we may form a correct opinion of the disease by pressure phenomena. Apart from a circumscribed *dulness* about the size of a silver dollar *laterally from the manubrium sterni* at the sterno-clavicular articulation, noted by some authors, especially upon swelling of the retro-sternal glands, which are often implicated, the central stock of bronchial glands which surround the bifurcation in the form of a heart, is of particular importance in the diagnosis. Here there may be an *intrascapular circumscribed area of dulness* at the height of the third to the fifth thoracic vertebra, together with increased *bronchial respiration*, or a distinct *stridor* with air hunger and dyspnea, thus betraying the presence of these glands. In like manner do the deep mediastinal glands which ascend as a part of the central stock of the bronchial glands, by surrounding and embedding the cava, the vagus and the recurrent laryngeal nerves, lead to cyanosis and edema of the face, or to periodic paroxysms of cough as in the case of per-



tussis, or, simulating a foreign body in the trachea, there may often be simultaneous asthmatic attacks, palpitation of the heart, paralysis of the vocal cord, etc. Where only a few of these symptoms appear, which do not permit of any other mode of origin, and are accompanied by constitutional phenomena, their presence is of great diagnostic importance, especially when the deep glands of the neck and subclavicular and intercostal glands are palpable, and I must emphasize the fact that by this means I have frequently made a correct diagnosis during the life of the patient.

**Pulmonary tuberculosis** in infants shows many peculiarities as compared with the same affection in adults. After puberty phthisis is the most frequent form of tuberculosis, the lung usually being primarily affected with the point of attack at the apex and predominated by miliary nodule formation. In the child pulmonary tuberculosis is less common than tuberculosis of the bronchial glands; it is *usually secondary*, beginning with glandular affection. The lower lobe is first attacked, the apex often remaining exempt. As in the bronchial glands here also caseation predominates; the formation of circumscribed inhibitive indurative masses which prevent the progress of the process does not occur or is incomplete. Pulmonary tuberculosis in the adult remains localized for a longer time. In the child the pleura and pericardium are implicated early; the appearance of miliary nodules with the formation of caseous masses in the visceral glands, liver, spleen, kidneys, is the rule.

In older children the affection of the apex is often the first demonstrable sign of tuberculosis, for here the condition is the same as in the adult in that the disease is not due to an obsolete tuberculous focus which has retained its virulence from infancy. *The apex of the lung is the point of least resistance* because of its slight respiratory excursus; the entrance of the bronchi of the apex into the main bronchus occurs in an obtuse angle, ventilation is difficult, and germs which have once found their way into this region do not easily escape; the tubercle bacillus therefore has time to accumulate here—in inoculation experiments it requires from ten to fourteen days—especially as the emptying of the pulmonary veins is difficult, the circulation inhibited—in a sedentary life there is even forced torsion—so that stagnation of the secretion is the natural consequence. In young children, particularly nurslings, the conditions are quite different. Here the heart dominates the thorax, the lungs are still deficient in breadth and develop outward anteriorly only with the increase of the osseous transverse diameter; the apices lack the fine branching of the bronchi, and the larger air canals are broad. Posteriorly the extensibility of the thoracic walls laterally to the vertebral column is slight, respiration is incomplete, and the secretory stagnation is influenced by the recumbent posture. It is true, in the first years of life the lower border of the thorax does not increase during inspiration, but lessens on account of the lack of power of the thin, yielding, elastic ribs to maintain an equilibrium with the marked tugging of the diaphragm, and this is all the more conspicuous in debilitated older children who are subject to catarrhal affections, infiltration and rachitis. Therefore the current of air which contains bacilli is directed to the glands near

the hilus and the bacilli embed themselves firmly in the pulmonary tissue directly in the broncho-pulmonary glands.

The distribution of tuberculosis from the bronchial gland occurs also by contiguity (*periglandular tuberculous pneumonia*), by way of the lymph tract (*peribronchitis caseosa, caseous pneumonia*), by way of the circulation, directly, by adhesion to the walls of the vessels, erosion, rupture into the lumen of the vessels (*infectious embolism*), or indirectly, by bacillary deposits in the thoracic duct (*hematogenous, localized, general miliary tuberculosis*). Thus, clinically as well as anatomically, we have *three well characterized forms of pulmonary tuberculosis: acute miliary tuberculosis, subacute caseous pneumonia, and chronic disseminated infiltration*. The principal representative of infantile pulmonary tuberculosis is caseous pneumonia, but the three forms present only gradual differences; they are often combined, or one form encroaches on another.

**Miliary Tuberculosis.**—Miliary tuberculosis occurs usually in connection with vascular tubercle. Cornet believes that the greater frequency of this form in young children is due to the fact that the bacterial toxins permeate the delicate vascular wall quicker, and are carried away more rapidly because of the active circulation, without leaving local irritative phenomena; obliteration and occlusion do not occur; the bacillus which follows, therefore, may rupture the delicate vascular wall, and this would happen all the more certainly if the starting-point were not so often in the gland, whereby to a certain degree, an encapsulation and retention of toxins is permitted. When, however, the invasion occurs by means of trauma, mechanical injury such as from whooping-cough, etc., the bacilli are enclosed in leukocytes under prurption of miliary nodules and rapidly deposited in all organs. If death occurs early we find fresh small nodules; if, however, the irruption occurs slowly, gradually, in exacerbations, the course is then protracted, and we find the nodules mostly undergoing caseation in the lungs, also in the visceral glands, the serous membranes, bone-marrow, meninges, etc. In the first three years of life often a few days suffice for the termination of this process; in older children the affection may last three or four weeks. Recovery is scarcely possible. The symptoms seldom give positive diagnostic support. Here also the general phenomena due to the toxins are the most prominent. Atypical high fever, occasionally sub-continuous temperature which ranges as high as 41° C. (105.8° F.), or the intermittent or the inverse type, or subnormal temperatures which continue for several days, always with a rapid pulse (180 per minute) and increased dyspneic respiration (40 to 60 per minute), stupor and apathy, indifference to the surroundings, rapid and extensive emaciation and complete exhaustion, enlargement of the spleen, petechiæ upon the skin, especially in the abdominal region, constitute the chief diagnostic criteria in contrast to the negative physical signs on the part of the lungs. The presence of bacilli in the blood and in the cerebrospinal fluid, and of miliary eruptions in the chorioid, is exceptional.

**Caseous Pneumonia.**—In contrast to the preceding affection, only rarely does the diagnosis of caseous pneumonia in children cause perplexity. The



demonstration of bacilli is not necessary for the recognition of this disease. Here it is particularly evident that physical diagnosis has not only lost nothing of its former importance but is of special value, for it is not only necessary to make a diagnosis, but to decide the stage of the disease—how far in each case the affection has advanced. We do not treat the disease, but the patient; “the individual must be kept within the horizon.” Usually we find general symptoms at the onset. No matter whether the disease appears after acute catarrh of the bronchi, after pneumonia, measles, whooping-cough, or otherwise, there has not been complete recovery from the preceding affection; the former health, color and activity of the child are lost, it tires readily, does not care to play, and becomes apathetic and melancholy. The increasing pallor, the poor appetite, the emaciation are unmistakable signs of retarded nutrition. Catarrh now appears with cough, as the result of a constant implication of the bronchial tree. This may for a time disappear, but recurs with renewed severity and becomes obstinate and persistent. The child complains of pain in the chest, which is increased by palpation; upon auscultation we hear pleuritic friction, rubbing, gurgling, or râles, distributed everywhere, above, below, upon the right, upon the left. The expectoration is profuse, the sputum purulent and blood-streaked. The respiration is rapid and dyspneic; subcrepitant râles become distinct; the dulness, which at first is limited, gains in extent; bronchial respiration and moist, crepitant râles persist in the same area which presents a tympanitic percussion note. Briefly, with tormenting cough, night sweats, hectic fever, diarrhea, and emaciation, the signs of increasing infiltration, or inflammation of the finest bronchi, of destruction, of cavity formation, become distinct. Even at this stage there may be an arrest of the process, especially in older children, if, similarly as in adults, there is circumscribed reactive peripheral inflammation with the formation of new connective tissue which leads to encapsulation of larger areas, with induration, contraction, and extensive bronchiectasis. The fever, which is due to toxins and entirely uninfluenced by drugs, may become less marked or disappear, provided the organism is able to produce bactericidal substances.

Nevertheless, the *phthisical habitus* becomes manifest. The expiratory position of the thorax with a vertical relation of the ribs, deepening of the intercostal spaces, the acute epigastric angle, and the flattened anterior surface, the retracted grooves at the neck and the smallness of all diameters, the decreased respiratory area, the winged scapulæ are all much more manifest than is usual in the adult. After a brief period of quiescence or apparent improvement the symptoms are exacerbated and, provided cardiac insufficiency or meningitis has not hastened the fatal issue, death approaches in a slow, dragging tempo, with the picture of toxin cachexia and usually without the formation of extensive new foci. The extreme pallor, the hectic fever, with changes of temperature from 34° to 40° C. (93.2°-104° F.) in a day, bilious vomiting, the colliquative intestinal symptoms with distended abdomen and effusion into the abdominal cavity, the edema of the extremities with obliterating thrombosis, the meningeal symptoms, occasionally with marantic cerebral sinus thrombosis,



the stigmata and petechiæ, the bed-sores, with rapid emaciation and atrophy which expose the contour of the skeleton, the prominence of the malar bones, the eyes deeply sunken in the hollowed orbits, the prominent clavicle and scapula, and spinous processes of the vertebra, and the distinct outline of the pelvis, conclude a scene of misery which can be better depicted with brush and palette than with pen.

**Tuberculous Meningitis.**—Tuberculosis of the lungs is usually a secondary affection in childhood—primary cases are extremely rare (Demme, Hammer, Wassermann, and others)—and usually develops from a focus in a bronchial gland. Quite the reverse, however, is the rule in tuberculous meningitis, the so-called basilar meningitis being particularly frequent in infancy in the proportion of about 60 per cent. to 8 per cent. in adults. The predisposition of the meninges to disease in infancy is due to the “affinity of the tissues for water,” whereby the meningeal mesh-work is constantly surrounded by a fluid rich in albumin and nutritive salts, which forms a particularly favorable culture medium for pathogenic organisms. Therefore in the first three years of life the brain increases to almost four times its weight at birth, while in the remaining years it enlarges barely one-sixth of its entire mass (at birth 13 to 14 per cent. of the total body weight, in adults only about 2.5 per cent.), which is proof of the enormous amount of labor produced by the increased metabolism and richness in blood. According to my experience no importance can be ascribed to heredity in the development of meningitis. Tuberculous meningitis owes its origin mostly to an old caseous focus in the bronchial glands (lungs, joints) from direct rupture into the circulation, as in the case of miliary tuberculosis; only in exceptional cases does it arise by contiguity from a neighboring caries of the bones of the skull (petrosal bone, the nose). In favor of the hematogenous development is the anatomical formation with its nodular swellings in the course of the vessels, and this also explains the simultaneous eruptions in other organs.

The *diagnosis of tuberculous meningitis* in adults is usually very difficult, provided lumbar puncture fails to reveal the true nature of the disease, but in infancy the symptoms are so typical and almost invariably recur with such unmistakable uniformity and regularity that, in the absence of other tuberculous phenomena, they might be designated as pathognomonic, and simultaneously permit a differentiation from meningitis simplex (convexitatis) and cerebrospinal meningitis. The individual symptoms do not arise suddenly and fulminantly, as in the former, with loss of consciousness, associated with delirium and general convulsions, or, as in the second affection, with chill, vertigo, vomiting, pain in the limbs, cutaneous hyperæsthesia, herpes facialis, enlargement of the spleen, and implication of the organs of sense, but the onset is gradual and insidious, corresponding to the exceedingly chronic infection and true to the anatomical localization and distribution of the process to the base of the brain. As in embolic processes due to organic disease of the heart, so here also does the hematogenous invasion follow the basal vessels to the bed of the *arteria fossæ Sylvii*. The gelatinous exudate, permeated with miliary tubercles,

surrounds the basal portions of the pons and chiasm in masses of varying size, forms anteriorly to the olfactory lobe, implicates the medulla oblongata and cerebellum posteriorly, and internally passes into the Sylvian fossa, follows all of the vessels, and is accompanied by detached or firmly grouped chains of transparent or opaque miliary tubercles which are not only found in the retiform tissue but also permeate the nerve substance, and even encroach on the trunk ganglia as far as the convexity. The ventricles are dilated and filled with exudate, the ependyma is granulated and thickened, the chorioid plexus is swollen and absolutely choked. Often, especially in protracted cases, as I have convinced myself, the membranes of the spinal cord are affected similarly as those of the brain.

Prior to the actual outbreak of the disease, as a rule, the discoloration of the skin, the deficient nutrition, and the increasing emaciation are conspicuous, especially upon the trunk and extremities, which present a distinct contrast to the round, full, bright face. The disease begins with severe spontaneous vomiting which may recur several times in the course of the affection; it is not attended with nausea, occurs independently of the ingestion of food, is usually forcible or projectile and appears suddenly and unexpectedly upon change of posture, or when sitting upright (*cerebral vomiting*). The pulse, the temperature, and the respiration, which at first were abnormal, fall to lower levels. Pressure of the basal exudate and the ventricular effusions cause irritation of the nerves at the base of the brain, particularly the pneumogastric. This explains the apparently altered respiration, which is slowed, deepened and interrupted. Frequently, after a brief arrest of respiration, there is a deep, sighing, labored breath, as though the child had a heavy weight upon its chest. The pulse decreases with the respiration and sometimes falls below 60 or even 50 per minute. The temperature also declines, so that in the three continuous curves of respiration, pulse and temperature there is quite a uniformity which, I agree with Heubner, is of great importance.

Pressure of the exudate upon the chiasm, etc., soon causes *ocular* symptoms. The eyes are rotated upward, frequently with their axes diverging, thus making more gruesome the peculiarly rigid and serious facial expression, and, with the furrowed forehead and the head bent backward, they lend to the countenance something superhuman, something outside our ken. Consciousness is usually retained. When sitting in bed, or when standing, there is a distinct sensation of vertigo. When the patients are recumbent and apathetic their arms are frequently crossed over their heads and the hands pick at the hair and lips. In consequence of collateral hyperemia a fleeting erythema frequently crosses the face which, like the other symptoms, such as slowing of the pulse, vomiting, headache, vertigo, is to be regarded as a *pressure phenomenon of the brain* due to limitation of space in the cranial cavity. The abdomen is retracted and there is obstinate constipation. Contraction of the muscles of the neck, retraction of the head, trismus, and gritting of the teeth are well marked, as in simple meningitis. With the ascent and with the increase of the exudate and the implication of the motor areas by irritation, torsion or compression of

the nerves upon their passage through the tense cerebral membranes, disturbances corresponding to the frequent change of irritation are noted, such as headache with convulsions, and facial paralysis which is usually partial. Strabismus, ptosis, contraction and distortion of the angles of the mouth also occur, with obliteration of the naso-labial fold—rudimentary, varying, fleeting—until finally, with the picture of *general paralysis*, and with a tired, worn, vacant facial expression and dulled consciousness, a rapid pulse, increased respiration and a renewed rise of temperature—occasionally with a brief return of consciousness which is often illusive—without a struggle, life quietly ebbs away. *Tuberculous meningitis* usually runs its course in fourteen days to three weeks; only rarely is there a deviation from the above type; then the onset is stormy, with fulminant symptoms and without essential prodromes, and the case terminates fatally in a few days; or, on the contrary, the disease may last for more than six weeks with focal symptoms such as occur in connection with tuberculosis of the brain—solitary tubercle in the cerebellum, trunk ganglia, medullary layer, cortex. In the latter condition, especially in older children, definite psychical depression becomes prominent: confusion, verbigeration, delirium—conditions of excitement which are intermittent and run their course with hallucinations, fear, speech disturbance, deafness, and blindness, and may then closely resemble the typical psychosis of adults.

**Intestinal Tuberculosis.**—While the forms of *infantile tuberculosis* heretofore described are distinguished by their frequency, as well as by their variation in typical form and course from the tuberculosis of later life, the *tuberculous diseases of other organs* are mostly secondary in this respect. This is especially true of the digestive tract. Rarely are the pharynx, soft palate, and uvula involved; the pharyngeal tonsil (adenoid vegetations), tonsils, tongue, etc., are seldom implicated (Piff, Gay, Siegert, Friedmann, and others), and even less commonly the stomach. The frequency of *tuberculosis of the intestinal tract* is also greatly exaggerated, as was mentioned in the description of nursing tuberculosis. There may be primarily a true “*feeding tuberculosis*” due to the ingestion of food which contains bacilli (milk of a phthisical mother or wet-nurse or of the cow, meat) or to the use of the same spoon or other eating utensil by a phthisical nurse; but much more frequently the disease arises secondarily by swallowed sputum which contains bacilli, or by autointoxication from some other organic focus by way of the lymph tract or circulation. With preference for the region of the ileocecal valve the infection is usually localized in the Peyer’s patches and solitary follicles after cessation of the miliary nodules, with infiltration and with crater-like, annular ulcers which girdle the transverse axis of the intestine, invade deeply to the serosa, and may lead to perforation likewise as in adults. In my cases which have come to autopsy a finding of this kind was rare. Anemia, emaciation, complete loss of appetite, often with burning thirst, atypical fever, severe peritoneal irritative phenomena, such as tearing colic with the diarrheal discharge of greenish, mucoid dejecta, or fatty stools of a light yellow, glistening, nodular appearance which



will not yield to any medication, are common recurrent symptoms, which, according to the intensity and extent of the local affection, may last for months or years and even until death, which, however, may occur surprisingly early from a generalized tuberculosis or after perforation due to peritonitis.

**Tuberculous Peritonitis.**—No matter whether tuberculous peritonitis be an accompanying phenomenon of general miliary tuberculosis without reaction, and of no interest clinically, or a circumscribed peritoneal tuberculosis originating from an intestinal ulcer, or a *chronic exudative* diffuse tuberculous peritonitis, the symptoms coincide with those of the same disease in later life. Pain upon pressure, spherical distention of the abdomen, particularly the umbilical region, palpable resistance and strand-like thickenings, or agglomerated tumors caused by the adhesion of intestinal loops, painful vomiting preceded by severe nausea, and clay-like, fatty stools may suggest the diagnosis. If recovery follows we may assume that we were dealing with a *non-tuberculous chronic peritonitis* such as has been described by Galvagni, Henoch, and others, or early laparotomy may have been responsible for this favorable termination. The explanation of this condition is disputed; whether the intra-abdominal decrease of pressure combined with improvement of the circulation, whether the neoplastic contracting cicatricial tissue, or the atmospheric air is the favorable factor, has not been determined. The elimination of pathogenic bacilli and their toxins, which results in the removal of the exudate, may play a large part in this, as also perhaps the reactive inflammation brought about by laparotomy. Ingianni has recently produced aseptic peritonitis in animals by the intraperitoneal injection of alcohol, which renders them immune to tuberculosis, and he regards the acute peritonitis and marked phagocytosis after operation as the cause of recovery from the disease.

**Tuberculosis of the Mesenteric Glands.**—This affection, sometimes called *tabes mesenterica*, which is often intimately related to tuberculosis of the intestine and peritoneum, frequently causes diagnostic perplexity from the fact that the enlargement of the glands can rarely be demonstrated by palpation. The mesenteric glands are attacked much less commonly than the bronchial glands, in the proportion of about 1 to 20.

**Tuberculosis of the liver, spleen, kidneys, genitalia, and the skin** presents no special peculiarities in children and therefore will not be discussed.

**Tuberculosis of the Bones and Joints.**—This localization of tuberculosis assumes such a predominant part by its frequency even in earliest childhood, and is of such essential surgical interest, that a special chapter will be devoted to it in the volume on surgery. I have already alluded to its relation to scrofula. Here it need only be emphasized that the implication of the bones and joints is often the *first manifestation of tuberculosis in infancy*, either primarily, which is rare, or secondarily by metastasis from an old focus, although among 67 cases of tuberculosis of the bones and joints. Orth was unable in 14 instances to find any originating focus.

In the three varieties, designated by König *granulation tuberculosis*, *tuber-*

*culous necrosis and infiltrated tuberculosis* the affection may occur in all bones of the skull, the face, the trunk, or the extremities, in isolated or multiple foci, although certain bones are especially prone to the disease, such as the long tubular bones and vertebral bodies. The affection often leads to rapid caseation, caries with softening, abscesses, or necrosis, with sequestration which, from its wedge shape has been alluded to by Volkmann and König as "*tuberculous infarct*" in proof of its embolic nature, which has also been verified experimentally by W. Müller. Apart from the great predisposition, which causes the rapid growth of bone and a particularly favorable circulatory relation, traumatic influences evidently play an important rôle in the frequency of bone and joint tuberculosis, since boys are affected twice as often as girls. Schüller, Müller, and Krause have confirmed this in their experiments. Krause assumes that slight injuries which produce no decided irritation or succeeding symptoms are more important causative factors than abrupt and severe injuries, because, during recovery from the latter, the reactive tissue formation is so energetic that the tubercle bacilli cannot withstand it. This conclusion coincides with the experimental finding that miliary tuberculosis occurs much more rarely in chronic, caseous, tuberculous pneumonia with connective tissue proliferation, under formation of indurative fibrous masses and retraction of tissue, than when tuberculosis runs its course in early life without these peripheral reactive inflammatory processes. When, as in spondylitis with simultaneous destruction of the ligamentous masses, there is an extreme tendency to suppuration, which represents the product of tuberculosis of the vertebra, the pus does not remain in the focus but wanders into strange territory as a gravitating, congestive abscess. This pus formation has nothing to do with gravitation but, independently of the law of gravity, follows certain tracts, which are defined by anatomical arrangement of the tissues, into a preëxisting space such as has been formed by interstices of the loose cellular tissue which unites or separates the organs, surrounds them, or permeates them. The distribution of the abscess depends upon its distensibility and regional arrangement—varying according to the localization in the cervical, thoracic or lumbar portion of the vertebral column. Similar conditions exist in tuberculosis of the joints, which is commonly propagated from the bone by rupture of an osseous focus in the synovial cavity (Volkmann), with exudation and tendency to destruction of the synovial membrane and caseation and disintegration of the cartilage of the joint. In the further course of the disease there is an involvement of the soft parts, with swelling and the formation of caseous granulation foci in the muscles and in the intramuscular and subcutaneous tissue, with numerous circumscribed or extensive abscesses and many entangled fistulous sinuses. Wherever the reactive inflammation tends to limitation, the new bone formation (osteosclerosis) and connective tissue proliferation permit healing, although usually with circumscribed defects and remaining deformities. Notwithstanding the limitation and encapsulation, naturally we have no assurance that quiescent germinating material does not remain.

Tuberculosis may involve *all joints*, but some articulations are especially



predisposed. In contrast to adults, in the child the hip, the knee, the foot, the elbow, in their order of frequency, are attacked, and here the osseous origin of the disease is especially prominent. Lannelongue asserts that among 100 cases of *tuberculous coxitis* about 70 per cent. occurred in the first ten years of life. The anatomical relations of the hip-joint, the processes of growth, the comparatively late ossification of the head of the bone, trochanter and pelvic floor, the intracapsular location of the neck of the bone, and the great circulatory richness of the joint give us an explanation of the overwhelming frequency of coxitis in youth. Karewski states that among 999 cases of infantile tuberculosis of the joint 365 cases, i. e., 37 per cent. were coxitis.

## PROPHYLAXIS AND TREATMENT

No matter in how far we acknowledge the relationship of scrofula and tuberculosis, they participate in a common *therapy*. The combat of those plagues which attack the marrow of the nation is to-day justly regarded in our civilized countries as one of the most important labors of state hygiene and social reform. From this standpoint, at the last Congress for Combating Tuberculosis as a National Disease special attention was devoted by the most eminent authorities to the value and benefit of *hospitals at springs and at the seashore* as a most *important prophylactic measure* for diseased children, and to the necessity of establishing *sanatoria* for the tuberculous, and *homes* for the non-tuberculous who are predisposed to this affection by their origin and surroundings. The constantly widening circle of this land plague has convinced both experts and the laity that notwithstanding all endeavor, study and investigation, we are unable successfully to combat tuberculosis with any remedy in our *materia medica*; but we have attained a clearer understanding of the manner of infection and have learned especially the sources of tuberculous infection in infancy, and now the battle must be waged anew with the reinforcement of *hygienic prophylaxis*. *Naturally, this must begin with infancy*, for here is the so-called "*family predisposition*," from which "*family infection*" (Heubner) originates. Notwithstanding the widely distributed educational effort, both spoken and written, in regard to the great danger of tuberculous infection, the restriction of the marriage of tuberculous persons will remain a *pium desiderium*, and the removal of the infant from tuberculous parents will not be accomplished usually from *inepta parentum charitas* (unprofitable parental love); but we must not cease to demand such a sacrifice and to see that it is carried out wherever possible. The least requirement is that the phthisical mother must not nurse her child but give it to a healthy wet-nurse. The infant should not share the same bed-room with the mother; it should be protected from kissing; the tasting of the child's food by the mother is to be prevented, and suspected brothers and sisters kept away. Tuberculous servants, nurses and governesses should be excluded from the house. Hygienic cleanliness of the child and its surroundings must be enforced as soon as possible. Atten-



tion should be devoted to the care of the skin, the mouth, the nose, the ears, the anus and genitalia; the nourishment of the child should be according to modern dietetic methods. *Milk*—the best exclusive or principal food of children in the first years of life—must be of unquestioned quality and preserved by boiling or sterilization in the house of those who consume it. This precaution is especially necessary, as the milk of tuberculous cows is the carrier of tubercle bacilli, and there is no law which restricts the sale of infected milk and milk products. According to Siedamgrotzky, Bang, Ebert, Jacob and other investigators, the result of the reaction of the tuberculin test in cattle is distressing, and shows the importance of the question for infancy in that the milk of cows that react to tuberculin is always suspicious. Bang has shown that the calf which is removed from the mother cow and fed upon boiled milk does not become infected but remains free of tuberculosis. Upon this fact, with the aid of the tuberculin test for diagnostic purposes a *systematic method of extinction* for the control of bovine tuberculosis is dependent.

Antituberculous prophylaxis, as already indicated, is not to be limited to infancy; a strict watch must be kept over the cribs and nurseries, kindergartens, play-rooms, schools, orphan asylums, hospitals, and other institutions, and a strict control of the health of those children who are sent to the country during the holidays is necessary for the protection of others who are well.

A proper nutritive therapy must be instituted in infancy in children who are suspected of being scrofulous or tuberculous, or who present manifest symptoms of tuberculosis. Here *the nutritive remedies are curative and the curative measures must be nutritive*. In this respect milk again occupies the first place. Milk foods, buttermilk, sour milk and cream with cacao, cacao to which some vegetable has been added, and barley coffee are excellent foods. Mucilaginous soups with bouillon and Leube's meat solution, or with boiled meat, preferably white meat, pigeon, chicken, veal in the form of purée, game, roast beef, boiled ham, tongue, and beef minced or chopped up with butter, with rye bread; at mid-day, after the meat, some young vegetables which does not cause fermentation, such as peas, beans or spinach, with potatoes, some light starchy food, and wine gelatine are permissible. *Sweets*, biscuits, cakes, chocolate, delicatessen, hard eggs, *wine*—particularly the sweet southern wine such as Malaga, Madeira, sherry and port wine, which often reach the consumer in very questionable quality—are *strictly prohibited*. We must not be content to prescribe the food under such general terms as "strengthening diet, etc.," but the quantity and quality and the time of meals should be given for the little patients in writing.

Special attention must be given to the salubrity of the dwelling, particularly of the bed-room. *Sunlight and air* are the greatest enemies of the bacillus; damp, dark, poorly ventilated living-rooms are its breeding places.

The important curative factors therefore are *sea-baths*, with sunshine, fresh air, and a salty ocean breeze which acts as a cleansing broom. But in the choice of a suitable region the general constitution and the individuality of the child must be considered.

The *Baltic Sea*, a closed basin which partially freezes, with a strong influx of cold, sweet, river water in the spring, and therefore with a less amount of salt and a lowered temperature, and without tide and waves, is serviceable in the late summer for *erethismic scrofulous children and those suspected of tuberculosis*; Cranz, Zoppot, Colberg, Misdroy, Swinemünde are the milder, Sassnitz, Binz, Doberan, Travemünde the more invigorating regions. Delicate, anemic and irritable children should at first take only warm sea-baths. The open *North Sea* with the warming influence of the Gulf Stream, a greater amount of salt, with its ebb and flood and larger waves, is more suitable for *torpid scrofulosis*. This affection requires greater warmth, more stimulation, and here the effect of a maritime climate is much more marked. Sylt, Wyk, Amrum, Langer Oog and Wangerog, Norderney, Yuist and Borkum, Scheveningen, Blankenberge, Ostende, are preferable according to the individual conditions. *Sanatoria and hospitals*, open throughout the year, must attain larger accommodations and better arrangements for bathing and for the aid of children in straitened circumstances. Patients of means may go to the beautiful winter stations on the Adriatic and the Riviera, or to the east coast of England.

In addition to sea-baths, brine-baths—often combined with the internal use of mineral spring waters which contain chlorid of sodium, iodin, or bromin—exert an excellent action in scrofula and tuberculosis, particularly when there are accompanying chronic affections of the skin, enlargement of the glands, inflammation of the bones and joints, with their sequelæ; in these conditions the brine-bath may be strengthened with lye. Erethismic natures require mild brine-baths, torpid temperaments the stronger baths. When there is a tendency to catarrh of the mucous membranes those brine-baths should be employed which include methods for graduating the strength of the brine. The number of brine-baths is enormous. Dürrenberg, Kösen, Salzungen, Hall, Hallein, Heilbrunn, Soden, Ischl, Aussee, Berchtesgaden, Reichenhall, Kreuznach, Krankenheil-Tölz, Nauheim, Oeynhausen are but a few.

The *symptomatic treatment* of scrofulosis and tuberculosis is naturally very important. In scrofulosis especially attention must be devoted to diseases of the skin and mucous membrane, since it is here that new germs constantly find ingress to the organism. In tuberculosis proper measures must be instituted in regard to expectoration, cough, hemoptysis, fever, vomiting and diarrhea.

Very little is to be expected of the so-called *specific general treatment*. The *preparations of iodin and iron* may offer valuable aid in certain cases, especially in scrofula. *Cod liver oil* requires an intact gastro-intestinal tract. In erethismic scrofula where metabolism and excitement are increased, the graceful structure and thin body require fat, and a methodic treatment with cod liver oil is in place. In the torpid form, with a flabby physique, constant digestive disturbances, retarded metabolism, and great repugnance to cod liver oil, nothing favorable can be accomplished with this remedy; on the contrary, the excretion of the sour, offensive feces which contain volatile fatty acids, is increased, the nutrition still more involved and digestion ruined. *Creasote* I believe to be a very valuable remedy, perhaps the only one which has retained confidence in the

treatment of tuberculosis. Given to small children, in water with aromatic tinctures, or with the addition of a few drops of ether, or in cod liver oil, to older children in gelatin capsules, it has produced excellent results in my hands. It should be instituted early and the dose should be gradually increased. Improvement of appetite and of metabolism are the first noticeable results. Albu and Weyl observe that even after prolonged administration of creasote virulent tubercle bacilli may still exist in the sputum. This should not prevent our use of the drug; in many cases, after a positive improvement in nutrition and of the general condition, I have observed a conspicuous decrease of bacilli in the expectoration, and I believe that its action is principally a chemical *immunization* by destruction of the toxins. Creasotal and thiocol appear to exert a similar effect.<sup>1</sup>

*Tuberculin* has been generally abandoned; it is of value for diagnostic purposes but it is not reliable nor without danger. Almost all authors, with the exception of Maragliano and Spengler, advise caution in its employment as a curative measure in tuberculosis, at least in childhood.

Although at this time we have no specific for tuberculosis we must not abandon hope. Perhaps *serum therapy* will here also disclose new ways and means. However, we are not helpless, but have many protective measures at our command against the most destructive of all plagues. Our present way lies in prophylaxis, hygiene and dietetics. Here, proceeding methodically, without weariness, we will slowly, very slowly, but nevertheless certainly, find the means to prevent this pest and to destroy the root of this poisonous growth.

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<sup>1</sup> Creasote holds an uncertain percentage of guaiacol. That is why I have preferred for nearly twenty years (upon the recommendations of Schüller) the use of guaiacol, or (the more tasteless, but more expensive) guaiacol carbonate. I never treat a chronic case of tuberculosis without it. As a tissue builder I give small doses of arsenic. I never omit giving a moderate dose of a cardiac stimulant when indicated. Small doses of digitalis may be given through months and years for that purpose. What I published on that subject 1884 (Trans. Med. So. St. New York) I have verified in thousands of cases since. There is no cumulation when thus used. As they have commenced to use digitalis in this way in Germany, in stray cases, for the same purpose and according to my rules, we Americans shall adopt the method.—EDITOR.



## HEREDITARY SYPHILIS

By H. FINKELSTEIN, BERLIN

As the entire subject of syphilis will be discussed in another portion of this work by an authority<sup>1</sup> I will content myself with a retrospect of the enormous number of clinical phenomena with which we meet in medical practice as the consequence of heredity.

The *history of syphilitic families* presents a scale of gradually decreasing effect upon the descendants. At first there are abortions; in later years premature labors. Then the period of pregnancy gradually approaches the normal, but the children are still-born, often a *fœtus sanguinolentus*. Living children are next brought into the world, at first greatly diseased, in whom life is soon extinguished; later more vigorous children are born who present the first consequences of a specific infection after some days. Finally, the power of heredity is exhausted and the children remain free of symptoms.

Not infrequently there are exceptions to this ordinary course. Even when no artificial interference has been attempted through the beneficent influence of energetic treatment, sometimes a child is born, among others who are extremely feeble or even dead, that is but slightly affected or perhaps perfectly healthy. It has occurred of twins that one was affected and the other was free of disease. Furthermore, recently infected parents may beget only healthy children. *The hereditary transmission is therefore not obligatory*, and many an experienced syphilographer believes that the importance, especially of spermatogenic heredity, has for the most part been grossly exaggerated.

Notwithstanding such deviations, our average experience may be generalized by certain conclusions which approximately are laws: *With the approach of the period of parental affection the deleterious importance increases for the offspring, i. e., the severity of infantile syphilis.* Again: *The more severe the disease in the child, the earlier in life are the symptoms unfolded.* Therefore, fetal and severe syphilis, and extrauterine and mild syphilis, on the whole become parallel conceptions, and the second again is the more benign the longer the interval from the day of birth to its appearance.

There is also an anatomic foundation for the greater deleterious effect of fetal syphilis in the form of extensive changes of vital organs, which usually do not occur when the disease begins extrauterine but are substituted by symptoms on the part of the skin and mucous membranes. Much labor has been expended

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<sup>1</sup> E. Lesser, "The Heredity of Syphilis." Volume on Dermatology.

for an explanation of the varied localization of early and late hereditary syphilis. Thus, Hochsinger believes that the virus has a special affinity for glandular organs and attacks them at a time when the greatest formative and nutritive stimulation exists in the region involved. In fetal life this aggression occurs in the internal organs; after birth in the skin, which is of vigorous growth. Nevertheless, late post-conceptional infection which takes place after the development of the internal organs is completed may be localized in the viscera (Finger), and eruptions are not unusual in the fetus. The central nervous system—not rarely involved during fetal life—is seldom attacked *after* birth, notwithstanding its enormous growth. Heubner's explanation is much simpler. He holds that an attenuated poison, although capable of adhering to the skin, cannot propagate where there is energetic circulation such as occurs in the internal organs. Therefore not the time but *the severity* of the infection is the determinate factor. In severe, virulent syphilis the internal organs are *always* affected, immaterial whether the outbreak occurred in fetal life or a few weeks after birth. Proofs of the latter condition are numerous. That such cases are seldom observed arises from the fact that only the rare coincidence of recent, severe, untreated eruptions with fatal complications brings such children to the autopsy table.

Therefore fetal syphilis is predominantly visceral, not because it is fetal, but for the simple reason that only a *severe* infection can produce the disease *in utero*; the syphilis of infancy runs essentially an external course because it is usually mild. When, exceptionally, the syphilis of this period is apparently severe, as is indicated by the intensity of the external symptoms, it is very probable that a visceral localization will also be found.

Notwithstanding these indefinite boundaries it is convenient, for purposes of discussion, to divide the affection into *fetal syphilis* and *syphilis of infants*.

## I. FETAL SYPHILIS

The factor which raises a suspicion of syphilis in the fetus is the *intra-uterine death with abortion or premature labor*, especially if repeated and other sufficient causes are not apparent. This suspicion can be confirmed only by an *examination of the internal organs*, except in those cases where there is pemphigus, which at once removes all doubt.

Histologic changes do not occur earlier than the beginning of the fifth month, and the cause of death of the embryo before that period must be sought elsewhere; either it is due to the *severity of the general intoxication* which destroys the delicate germ, or *disease of the placenta* gives rise, through changes in the vessels, proliferative granulations, gummata, induration and contraction in the villi, to destruction and constriction of the blood lacunæ and retarded metabolism, the result being the death of the fetus.

*Definite changes in the organs* may be demonstrated from the fifth month.

The recognition of these changes frequently presents no difficulty, espe-

cially when they appear in various regions as *large gummata* or *gummatous infiltrations*. In the overwhelming majority of cases, however, the *macroscopic signs are less obvious* and are limited to a change in the color and condition of the blood, and to an increase of volume and consistence of the affected parts, the specific foundation of which is only revealed by the microscope.

*Enlargement of the spleen* and the frequent *volumetric increase* of the liver are of diagnostic significance, and in proportion to the weight of the child are very decided (liver 1:14.7, spleen 1:198; in contrast with 1:21.5 and 1:325 respectively of the normal. All uncertainty will at once be removed by an incision through the epiphysis of a tubular bone to determine the presence or absence of the *osteocondritis of Wegner*, which is an almost constant pathognomonic sign of the disease.

The essential feature of fetal syphilis is the specific disturbance of endochondral ossification. *The calcification of cartilage continues undisturbed, but resorption is prolonged and change into bone does not occur*. The proliferating osteogenic tissue between the column cells of the cartilage is denuded of osteoblasts and resembles a simple granulation tissue. This explains the *macroscopic appearance of the cartilagino-osseous border*: distribution of the whitish, mortar-like zone of provisional calcification, serrated limitation in consequence of prolonged existence of the calcified column cells and irregularities in the prominence of the granulation tissue, sclerosis and friability of the entire bone, because everywhere the deposits of calcium are loosened incompletely. In later stages, under the influence of the specific damage, there may be fatty degeneration, liquefaction and necrosis, and, finally, solution of the feebly elastic calcium structure of the cartilaginous epiphysis.

Further diagnostic criteria are furnished by *microscopic examination of the internal organs*, particularly the kidney, spleen, thymus gland (Hecker).

The changes peculiar to hereditary syphilis may in general be defined as *small-cell infiltration, associated with proliferative phenomena in the connective tissue, which is focal or distributed diffusely over entire organs or portions of organs, and possesses a conspicuous tendency to indurate*. *Typical vascular disease is also observed*.

The individual components of this process are: 1. Multiple circumscribed *cell accumulations*, as well as intraparenchymatous, interstitial, and perivascular, which may attain a macroscopic prominence (*miliary syphilomata*) and lead to gummatous processes when especially localized around the larger vessels. They are pathognomonic of syphilis. 2. *Diffuse and circumscribed connective-tissue proliferation*, which may increase to the point of induration and cirrhosis, undoubtedly is also of great diagnostic importance.

3 and 4. The *diffuse, small-cell infiltration* and peculiar *epithelial structure* which are found in the lungs, kidneys, and thymus, are more difficult to estimate. The *diffuse, small-cell infiltration* often permeates the tissue to such an extent that at first glance the structure is not recognizable. Its specific importance has recently been questioned, and Hecker, Terrier and Karvonen in particular have emphasized the fact that even under normal conditions



the fetal organs, notably the liver, may contain a diffuse accumulation of cells and thus cause confusion. It is likely that this variety of microscopic pictures does not present, wholly at least, a specific inflammatory proliferation but is only a fetal phenomenon, perhaps of a somewhat longer existence under the influence of syphilis, but otherwise normal.

*Epithelial structures*, in the form of focal accumulations of cylindrical or cubic epithelial cells or persistent cylinders, are found in the *lungs* as the remains of fetal pulmonary tissue. In the *kidney* there is a subcapsular neogenic zone in which the canal system also remains in a fetal condition, without separation into Bowman's capsule, convoluted tubules, etc. Hassal's thymus bodies will be mentioned later. The explanation which has been given for these conditions is that they are the remains of an abnormal separation of portions of glands which has arisen from a syphilitic inhibition of growth. Hochsinger believes them to be due to the local damaging effect of the specific proliferation; Karvonen ascribes them to the general effect of toxins.

Karvonen's theory endeavors to unite all of these points of view. It contrasts a syphilo-toxic influence, which inhibits development and causes diffuse cellular proliferation and persistent epithelial formation, with circumscribed specific proliferation. The inhibitive influence is shown by the persistence of the tissue in the fetal condition: The milder grades of perivascular infiltration are only normal vessels about to be formed; the diffuse cellular infiltration is in fact a mesenchyma which remains in an embryonal state, and the epithelial structures are to be considered likewise. This, then, is the basis upon which the typical focal proliferations develop. Although certain authorities (Hecker) have contradicted some of these points it cannot be denied that the theory has much to commend it. In favor of an existing influence which inhibits development is the generally protracted growth of the syphilitic fetus and primarily the process, above described, in the epiphysis of the bones. The theory of Karvonen combines many of these well known facts under a general law.

In regard to the individual organs, the *infiltration* and the newly formed *connective tissue* along the portal capillaries permeate the *liver* between the trabeculae and in the further course may lead to *diffuse sclerosis*. The capsule is often decidedly thickened. *Miliary syphilomata* are common; rarely there are larger nodules and gummatous inflammation around the porta which may originate from the biliary passages as well as from the trunk of the portal vein (peripylephlebitis, Schüppel).

The *pancreas* is a selective area of infiltration and induration. Recent investigators believe that the *kidneys* also are constantly involved, especially by cellular infiltration around the smallest cortical vessels.

Indurative enlargement of the *spleen*, as an important and almost pathognomonic symptom, has already been mentioned.

In the *lungs*, apart from large or smaller gummatous nodules, "*white pneumonia*" is to be found: blocking of the alveoli and bronchi with fatty degenerated round and epithelial cells, with unchanged interstitia. *Interstitial*

(diffuse infiltrative) *pneumonia* is more frequent and is commonly associated with white inflammation which in pure condition is extraordinarily rare.

The *thymus gland*, which in addition to diffuse infiltration may contain nodules, occasionally reveals the rare finding of cystic spaces filled with purulent masses (Dubois's disease). This condition is due to an inhibition of involution, in that the remains of the epithelial tubules which form the first foundation of the gland (Hassal's bodies) do not disappear but become distended and enlarged by the entrance of thymus substance or of thymus cells which afterward undergo fatty degeneration.

Relatively frequent characteristic changes have been found in the *central nervous system*, especially in recent years (Jürgens, Matthewson, Gasne, Gilles de la Tourette). Other organs which have not been especially mentioned may also be the occasional seat of pathologic products.

Vascular proliferation and perivascular infiltration are occasionally observed in the *umbilical cord* and may sometimes be of diagnostic value when autopsy is impossible.

It is obvious that more decided grades of the visceral changes which have been described are not compatible with life. Therefore the frequency of stillbirths in the anamnesis is important in connection with the next category, the main factor of which is the "polymortality" (habitual mortality) in syphilitic families. It is true, other children *may be born alive but prematurely, as wizened and sickly subjects*. Significant of these syphilitic premature births is the *retardation in weight and bodily development*, in contrast with normal children carried the same length of time, and a "*defective vitality*" which cannot be combated by the most careful nursing and which is manifested by a non-resistance to even secondary infections. Many die after hours or days; only a small minority live more than a week.

The last group is of greatest interest to the physician. These children also, *carried to full term, but born with the florid symptoms of the disease*, must be regarded as fetal syphilitics.

Here also are found a *shortage in weight*, sometimes amounting to actual atrophy, and the "defective vitality" which permit the assumption of severe internal localization. Among the occasional recognizable external signs is *rhinitis*, which later becomes so important, and exceptionally a *bullous exanthem*—*pemphigus syphiliticus*—is observed.

The exanthem is first distributed in the form of scales over the entire body, soon followed by the formation of a serous fluid, afterwards purulent, resembling vesicles, which raises the epidermis. An important factor in the differentiation from pemphigus neonatorum is the localization upon the *palm of the hand and sole of the foot*, for here the largest vesicles are situated, sometimes of the size of a cherry. Extensive and severe cutaneous affections may arise by coalescence, formation of fissures, and ulceration.

The *prognosis* of these cases is bad,<sup>1</sup> not so much because of the peculiarity

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<sup>1</sup> In regard to the necessary strict differentiation from a pustulous syphilide, see later.

of the eruption, although it resembles syphilis maligna in its resistance to specific remedies, but on account of the serious internal complications. Only the greatest care will prevent secondary septic infection.

## II. SYPHILIS OF NURSLINGS

### A. SYPHILIS OF THE SKIN AND MUCOUS MEMBRANES OF THE FIRST ERUPTIVE PERIOD

A child, apparently normal, is born and for a time a slumbering affection is unsuspected, until the appearance, often surprising, of unquestioned symptoms reveals the true state of affairs. This *period of latency* may be measured by days, weeks, or months. Among 344 children Rosen saw the infection appear in the first month 157 times, in the second month 111 times, in the third month 42 times, and much later in 31 cases. In 46 of my patients the earliest appearance of the exanthem was as follows: between the first and third days 4 cases, end of the first week 5, second week 4, third week 5, fourth week 9, fifth week 5, sixth week 3, seventh week 7, and 1 case each in the eighth, tenth, and thirteenth weeks and fourth month. No trustworthy reports can be obtained of the beginning of the coryza which for some time precedes this condition.

The existence of such an "*incubation*" period is of the greatest practical importance. The suggestion is sufficient of the serious conditions which may be produced if a child, who has been given to a wet-nurse, subsequently becomes ill, or if, after some weeks, the child of a wet-nurse develops syphilis. Directors of foundling and orphan asylums must at all times bear in mind that an apparently healthy child may be the source of contagion to its adopted parents.

Therefore the earliest possible diagnosis, the recognition and scrupulous observation of the first symptoms of the disease, are essential. The secondary period of hereditary syphilis begins with an exanthem only in the minority of cases, just as in an acquired syphilis. As a rule there are insignificant antecedent phenomena, *often for a long time previously*, which at least arouse suspicion and frequently give to the experienced physician positive proof of the existence of the disease. Their consideration is all the more urgent since transmission to those about the patient is possible from these early phenomena.

Primary among these symptoms, and one which is seldom absent, is *snuffling*, at first dry, later accompanied by mucous râles—a sound during nasal respiration which is particularly distinct when the mouth is closed. The underlying cause is *coryza syphilitica*, which begins with dry swelling, succeeded by a tenacious, crust-like secretion, and finally shows a profuse purulent and hemorrhagic discharge, probably due to secondary bacterial accumulation, with simultaneous infiltration, fissures and ulceration upon the alæ of the nose. If the nasal passages are entirely blocked there is a conspicuous position, particularly of the neck, which resembles opisthotonos and probably facilitates



the introduction of air. With an intense and prolonged coryza the pathologic processes exceed the limit of the mucous membrane and lead to changes *which may also affect the external appearance of the nose*. In mild cases the change is limited to a cicatricial retraction resembling pug-nose, and an occasional contraction which may occlude the nasal passages almost to the extent of impermeability. Involvement of the cartilaginous and osseous structures (inflammatory nutritive disturbances, rarefaction or softening in the growing cartilage and bone) results in a sinking of the bridge of the nose (saddle-nose). Ulcers, perforations of the septum, and gummatous destruction of bone are exceptional occurrences. Perforation of the hard palate has been reported.

The "micropolyadenia" (multitude of small glands) of acquired syphilis is usually absent, and is of no diagnostic value should it occur. Even the enlargement of the cubital glands, which some authors declare to be important, in my experience is significant only in combination with other suspicious symptoms and when the glands are prominent (the size of a pea or larger). Otherwise slight enlargement is common. On the other hand I have not thus far found a chain of palpable glands along the sulcus bicipitalis elsewhere than in syphilis, although only in older infants. The *splenic enlargement* can be utilized in diagnosis only in conjunction with other phenomena.

Among 52 children I found a palpable spleen in 39 cases (75 per cent.); of these 4 attained the size of a decided tumor. The importance of this finding is lessened by the fact that at this period particularly a slight enlargement of the spleen can be demonstrated in many non-syphilitics.

More typical are the cutaneous changes, especially in the face. Here we do not search for *pallor*—which has often been designated as characteristic, but which I have frequently not found—but for certain *peculiar phenomena of infiltration* which, as a rule, soon follow the coryza. Around the border of the lips a reddish yellow, glistening zone appears, several millimeters broad and somewhat coarse, which is soon deeply fissured and may even implicate the red of the lips. The condition around the ala nasi has already been mentioned. Even the region of the eyebrows is coarse, thickened, and slightly desquamated. In addition the face presents a brownish yellow or pale yellow color—as of "*café au lait* or of the fingers of a cigarette smoker" (Trousseau)—which assumes a red tint around the chin and is simply a milder form of the same infiltration. Similar changes may appear around the *anus, genitalia, and the flexor surface of the thighs*. At this time *alopecia*—scant hair upon the head, brows, and eyelids—becomes conspicuous. To summarize: the complexion, lymphatic enlargement, alopecia, coryza, snuffling, and the nasal quality of the voice present in typical cases an unmistakable picture, which, however, is readily recognized by experts in a less marked or rudimentary form.

Other symptoms referable to the skin need not necessarily appear in addition to the changes already mentioned. Nevertheless, with a typical course, cutaneous eruptions are to be expected in the form of *diffuse* or *circumscribed exanthemata*.

The *diffuse exanthemata* are localized preferably in the regions, already

mentioned, of the cutaneous changes and infiltration, and may be regarded as an exaggerated form of the same conditions. The swelling becomes more distinct and extensive, the color a brownish red, and the desquamation more marked, especially around the nose and mouth where the tense borders are permeated by radial fissures, and upon the ears, the forehead and the hairy scalp. The desquamation may increase to a true *seborrhea* which covers the surface with a scaly *rupial or eczematous crust*.

This "diffuse infiltration," as it is designated by Hochsinger, is said by him to involve extensive areas, especially the posterior surface of the lower half of the body from the sacrum to the heels, the anterior surface remaining exempt. Complications with macerating processes then produce an extensive intertrigo which corresponds in its location to "the leather patch of riding breeches." The specific importance of this form of eruption is probably not applicable to all cases.

The same infiltration appears on the *soles of the feet* either in the form of swelling of the skin, which becomes coarse and glistening, with folds and scales and of a light brownish yellow color, or as a scaly, bluish red erythema resembling lacquer. The palm of the hand presents the same appearance. The ordinary reddening of the planta, observed in atrophic children, is to be differentiated from that of a syphilitic affection by the thickness of the skin.

Diffuse exanthemata are a frequent but by no means constant phenomenon of the disease. Like the milder cutaneous infiltrations, they may be absent. Cases sometimes occur without the appearance of rhinitis, the development then being sudden, with an eruption, corresponding to its onset in adults. I have particularly noted the absence of all prodromal symptoms in cases where reasons on the part of the parents made a careful observation of the newborn imperative.

The characteristic *circumscribed exanthemata* develop either upon and between the diffusely affected areas or as primary cutaneous eruptions. Their appearance may be ushered in by fever, likewise as in adults. They require no comprehensive description; it is sufficient to call attention to the conformity with the picture of later life, to which but a few remarks need be added regarding special conditions.

True to the experience that the severity of infantile syphilis constantly diminishes with time, here also the forms which arise early are the more grave. Actual primary ulcerous exanthemata appear to be absent.

Thus, the *pustulous syphilide* only appears at the onset, seldom after the third week. The large pustules abound on the palm of the hand and sole of the foot, as in congenital pemphigus, which is in fact a more severe type. The unfavorable prognosis of congenital pemphigus, however, does not apply to the pustulous form—all of my cases were cured, and the diversity of opinion of various authors regarding the prognosis of pemphigus is probably due to the less distinct differentiation from the pustulous syphilide after the first or second day. According to my experience only those cases are serious in which not only the palmar and plantar surfaces and their surroundings are involved

but actual vesicles appear over the entire body. Papulo-pustulous efflorescences are significant of a more favorable condition.

The *exanthemata* of longer incubation are the *macular*, *papular*, and more or less *squamous* forms, and those resembling *roseola*. They may appear in profuse crops like measles, or as a few faint efflorescences. Larger plaques may develop by confluence. The preferred and characteristic localizations are on the palms and soles, where the desquamating forms, known as *psoriasis palmaris* and *psoriasis plantaris*, predominate. Scaly and often round infiltrates are found upon the fingers and toes; *paronychia* and *trophic changes* in the nails are common.

It appears worthy of remark that *mucous membrane plaques* and *condylomata* occur in the nursling during the first period of eruption—I have seen them upon the tongue, lips, pharynx, and genitalia—but they play no prominent rôle. In fact an implication of other mucous membranes than that of the nose is quite uncommon. Perhaps the most frequent involvement of the mucosa is in the *larynx*. Disease of this organ is characterized by decided *hoarseness* which may exceptionally increase to *aphonia*. The anatomical basis is probably the same as that of coryza. At the autopsy of one of my cases in which the aphonia had existed from birth the vocal cords were found covered with massive fimbriated papillomata; no other specific laryngeal changes were observed. In this early period severe and even fatal disturbances occasionally occur; stenosis with ulcerations and perichondritis, requiring tracheotomy, have been reported. A careful analysis of most of these cases, however, raises the suspicion that there were complications with diphtheria or sepsis.

#### B. SYPHILIS OF THE BONES AND INTERNAL ORGANS IN THE FIRST PERIOD OF ERUPTION

In the minority of syphilitic infants there is an implication of the same organs which are commonly attacked *in utero*. These visceral forms may be classified under *two groups*: first, that form in which beginning changes do not necessarily lead to death of the fetus, but continue for some time after birth ("projection of fetal syphilis"); second, those cases in which the internal organs present disease simultaneously with severe cutaneous syphilitic eruptions.

Relatively the most frequent are *disease of the bones*. Aside from the *gummatous thickenings* upon the internal and external surface of the flat bones of the skull, and the multiple *phalangitis* upon the fingers and toes which resembles *spina ventosa*, we are most interested in the condition of the long tabular bones.

Wegner's osteochondritis, which is of sufficient frequency in the nursling to maintain an anatomico-diagnostic importance at the autopsy, is not clinically recognizable in the mild and medium stages, except perhaps by the Röntgen rays (Holzknecht). Only when the inflammatory process attacks the periosteum are external changes demonstrable in the form of *spindle-shaped protuberances limited to the epiphyseal regions*, the anatomical foundation of which,



in the severer grades of the epiphyseal affection, is a many-layered thickening of the periosteum with a gelatinous or caseous infiltration. Such an enlargement may be found unilaterally or bilaterally upon all of the long bones, but seldom upon the trunk. The upper extremity is involved to a greater extent than the lower, therefore the affection can be best studied in the arms.

Here the thickening occurs near the elbow and frequently upon the distal epiphysis of the lower arm. The upper humeral epiphysis is rarely involved to a perceptible extent. While the *joints are yet intact* there is frequently a *swelling of the soft parts* and a *manifestation of pain* upon contact or passive movement. The most remarkable, though not obligatory, accompanying phenomena are peculiar *disturbances of movement*, which, following the example of Parrot, have been designated "*pseudo-paralyses*," and produce the following clinical pictures:

One arm or both are extended in *flaccid paralysis* and rotated inward with the pronated hand alongside the trunk. The position has a surprising resemblance to complete plexus paralysis. The hand when raised, has a pendulous motion with the position of radial paralysis. Movement is possible only in the fingers, although a tendency to muscle action is occasionally observed in other regions, notably the adductors.

The paralysis is not always so complete. Sometimes there is *only a difficulty of movement*. I have also noted a clinical picture which resembled paralysis of a peripheral nerve rather than of the plexus; for instance, in one case, a typical *radial paralysis*, in another a left-sided *claw position and failure of all movements dependent upon the ulnar nerve*.

The *pathogenesis of this motor disturbance* has not been satisfactorily solved. Some of the attempted explanations—and particularly that of Parrot—make it *dependent upon disease of the bone*. This leads to *solution of the epiphysis*, and the result thereof and of the pain on movement is a *reflex position of rest*. But in many of the cases the loosening, which is demonstrated by crepitation and abnormal movability, is not present.

Oberwarth maintains that the slightest change in the epiphysis produces a *periosteal irritation* whereby every contraction of the muscle causes pain and discomfort and for this reason there is flaccidity. By a similar train of thought Hochsinger assumes a *specific disease of the muscular insertion on the bone*.

These explanations may suffice for a number of cases, but not all, particularly when there is complete absence of pain upon passive movement and pressure, as is not infrequent. Further, there is by no means a direct parallelism between the osseous process and the disturbance in function. In cases of equal enlargement on both sides Hensch, as well as I, have noted only a unilateral motor disturbance. The rapid return of function under treatment, even *before* a visible influence on the bone, is likewise in evidence.

Accordingly, mention must be made of those views which refer the condition to *disease of the nerves*. Affection of the peripheral neuron, through the irritation of gummatous bony enlargements on the arm or in the intervertebral spaces (Reuter), is unlikely, as such a condition could scarcely occur without

marked sensory irritative phenomena, which are not clinically apparent. Meningeal or central processes, however, may be operative. In these cases a closer observation of the pathologic findings is necessary.

The purely nervous foundation cannot be rejected when there is *paralysis without a clinically demonstrable affection of the bones*.

The knowledge of this particular form of syphilitic paralysis is not extensive, nevertheless it is of great importance, as errors in diagnosis may lead to therapeutic sins of omission. Diagnostic confusion occurs all the more readily because the condition may be congenital, resembling obstetric paralysis, or, if the paralysis occurs after birth, often quite acutely, it has an apoplectiform appearance. Furthermore, this type of syphilitic paralysis is rarely accompanied by eruptive phenomena. Much more frequently one condition succeeds the other, or eruptions are altogether absent and only visceral complications indicate the etiology. For an understanding of this peculiarity the same remarks are applicable which will be found in the later discussion of syphilis of the liver.

*Clinically* these paralyses throughout follow the type of obstetrical paralysis. The most common form is complete lower plexus paralysis; paralysis of one upper extremity is unusual. The oculo-pupillary phenomena of Klumpke's paralysis (myosis, contraction of the palpebral fissure) may be present.

Two proofs are known at this time which favor the view held by Peters of a *central origin* of these paralyses. Zappert found meningitis in the cervical cord with columnar degeneration; in a case characterized by bilateral paralysis of the arm, paralysis of the sphincter, and flexor contracture of the leg, I found recent, partly diffuse, partly nodular, gummatous infiltrations of the entire central nervous system with here and there a well-developed sclerosis.

At this point must be mentioned the *contractures* of syphilis which occur alone or as the accompaniment of paralysis of the muscles of the neck, of the iliopsoas, of the flexors of the arms, etc., and which disappear under specific treatment. They are little understood and it is difficult to determine in how far specific *diseases of the nervous system*, perhaps also of the muscles, and further the *non-specific permanent contractures* which are so pronounced in very sick children, deserve etiologic consideration.

*In the legs* the same *osseous changes* and flaccid *paralyses* occur as are found in the arms, though the paralysis is much less frequent.

*Implication of the central nervous system*, as has already been mentioned, is not unusual in fetal life. It appears, however, that florid stages of *infiltration, sclerosis, meningitis, and vascular disease*, originating at this period, as occurred in my case just quoted, are rarely consistent with prolonged extra uterine existence. Nevertheless, it is likely that in the stage of cicatrization they give rise in a certain percentage of cases to *Little's disease* and other forms of *infantile cerebral paralysis*, as well as to congenital *idiocy* and *epilepsy*.

*Congenital hydrocephalus* also is frequently associated with syphilitic heredity, even such forms as are not influenced by specific treatment. In these cases the relation to syphilis resembles that in *tabes dorsalis*. Indications of

hydrocephalus (prominence of the fontanelles, downward glance), as Oedman-son rightly emphasizes, are common. But there is also quite a series of reported observations of well-marked cases which have been cured by an energetic administration of iodine and mercury; probably in these patients there was not an ordinary hydrocephalus but a *specific meningitis with marked exudation or with occlusion of the ventricles*, as has been described by Astros, Haushalter and Thiry, and was observed also in a case of my own.

In addition to that form of meningitis distinguished by ventricular effusion there is also a very characteristic gummatous *basilar meningitis* (Gowers, Hadden, Oppenheim) with special implication of the posterior cranial fossa, or occasionally, as in one of my cases, with involvement of the middle groove and defined by various symptoms on the part of the basal nerves. *Syphilomata* and *endarteritis* appear to belong only to the period of relapse.

*Disease of the eyes* may to a certain extent be regarded as a continuation of the cerebral affection. Among the most prominent findings is that of *gray* (post-neuritic?) *atrophy*, which runs its course without other concomitant nervous phenomena and is only discovered by special investigation in that direction (Kalischer). The fate of such cases is unknown. *Choroiditis* is apparently more common in later months. *Keratitis parenchymatosa* of the external eye is rarely observed in after life. *Primary plastic iritis* is pathognomonic and may develop *in utero*—I myself have found an extensive posterior congenital synechia in the newborn.

*Syphilis of the liver* in the form of tumescent *hepatic gummata* is of very exceptional occurrence in the nursling. More common is a *diffuse affection* which is rare in the adult. This is observed either as a recent *infiltration proceeding from the capillaries of the portal vein*, which results in a *portal hyperplastic cirrhosis*, or as the *biliary, icteroid form*, which is produced by changes in the bile-channel system.

*Recent diffuse hepatitis* as a complication of external eruptions is to be expected with greater likelihood the more the intensity of the external symptoms favors the severity of the individual case. *This condition can be established with certainty if, in addition to other syphilitic phenomena, an enlargement and especially a positive hardening of the organ can be demonstrated by palpation.* There is also splenic tumor. Jaundice and ascites are absent.

A positive diagnosis in those cases which have not yet reached the stage of sclerosis, and therewith an opinion in regard to the frequency of such cases in the living, is subject to many clinical and anatomical uncertainties.

Even the demonstration of an enlargement is not simple, except in extreme cases. Quite frequently in the normal infant the liver is palpated in the mammary line about 3 cm. below the arch of the ribs; therefore only greater deviations than this are of diagnostic importance. Within this limit, among 40 syphilitics I found hepatic enlargement in only 5 cases (12.5 per cent.)—greater than Kraus's figures (1.8 per cent.), but much less than Hochsinger's (31 per cent.). Furthermore, enlargement of the organ may not occur even in a severe affection.



In the absence of other symptoms, to consider the enlargement as a manifestation of specific hepatitis is not justifiable. There may be fatty liver, stasis, and other conditions. The result of anatomical investigation must also be utilized with caution. In addition to the delusive possibilities already mentioned, i. e., the relation of fetal conditions to those of life, there are disturbances such as gastro-enteritis, sepsis and other fatal complications, which may cause an interstitial accumulation of cells. We may assume with some certainty the diffuse infiltrating form of hepatic syphilis when we can demonstrate clinically a distinct increase in consistence.

When the disease of the liver has reached its acme in fetal life the child is born with an organ which is already in the stage of induration—with *complete cirrhosis*—and usually in a hyperplastic condition; exceptionally there is beginning contraction. Such patients are extraordinarily pale and cachectic, with distended abdomen over which the veins are plainly visible, often with dropsical symptoms, and above all with a conspicuously hard liver, usually still enlarged, as well as a coarse splenic tumor. As a rule there is a large amount of indican in the urine. Jaundice is absent in these cases; in a few instances ascites has been observed. Secondary symptoms of the disease are seldom noted.

This absence of exanthemata or of rhinitis does not justify a doubt of the specific nature of the cirrhosis. Anyone who questions it has forgotten that in these cases the period of eruption, and with it the time at which external manifestations are to be expected, was passed in the uterus, that traces may remain of a profound hepatitis in the form of induration, but not of a transitory and insignificant cutaneous affection. Theoretically we must expect only such changes as are equivalent to the hepatic affection; if cutaneous symptoms appear, as in a case of Henoch's, these must be explained as a relapse. Twice among 4 cases I have seen osteochondritis as an accompaniment, in one case induration of the testicles, and in the remaining case an isolated, severe, large lamellar psoriasis plantaris et palmaris appeared, as might be expected, weeks after the congenital hepatic tumor had been determined. The absence of simultaneous syphilides on the skin and mucous membrane is therefore no reason to reject the specific nature of the affection; instead it must be expected.

That cirrhosis is not observed more frequently and is only rarely combined with ascites is probably due to the fact that such severe changes seldom permit of a prolonged extra-uterine existence, and the few who survive these changes perish while there is yet compensation for the circulatory disturbance. In the infant there is an especial tendency to compensatory hypertrophy.

Enlargement due to simple diffuse infiltrations appears to respond readily to *therapeutic* measures. More tenacious, and therefore obviously of less favorable prognosis is the second category, although such cases are occasionally amenable to early and prolonged treatment. A case of Depasse's which was associated with ascites and several times required paracentesis, showed improvement in the course of two years; nevertheless a certain degree of enlargement and hardening persisted.

The second group—icteroid cirrhosis—includes a series of occurrences,

also rare, in which—usually directly connected with *icterus neonatorum*, more rarely separated from it by a certain interval of time—*high-graded icterus* develops, *sooner or later associated with acholic feces*. *The liver is large and hard*; there is *splenic tumor*. In pure cases ascites is absent. If the cases are grouped into a common picture the result is an occlusion of bile from the intestine which is complete at birth or has gradually developed in the first months after birth, and is associated with cirrhosis of the hepatic tissue. It is remarkable that in children who suffer to such an extent other symptoms of syphilis are not present or are at most merely suspected. The condition has frequently been observed in several children of the same parents.

The changes, the end product of which has just been described, pass their florid stage *in utero*, the same as portal cirrhosis. Chiari and Beck have found in still-born children and in those who have lived for a few weeks a *gummatous inflammation of the large biliary passages* which permeated the liver tissue, and in the process of new connective tissue formation and indurative contraction, necessarily led to changes in the form of obliteration of the biliary passages, cicatricial constricting proliferation, diffuse biliary (cholangitic) cirrhosis, and the effects of biliary stasis. At the autopsies of children who have succumbed after several months the anatomical picture shows the termination of these processes distinctly (indurative changes at the porta, obliteration to total disappearance of the biliary passages, cholangitic cirrhosis), while those cases in which bile is retained in the feces have not yet reached this stage.

Schüppel has described cases with *icterus*, *acholic feces*, and *ascites* in the fetus and in still-born children and their cause—in contrast to cholangitis in the preceding cases—he has recognized as a *peripylephlebitis*: a fibro-caseous inflammation starting from the main trunk of the portal vein which produces constriction and obliteration and in its further course may involve the biliary passages. *Clinically*, in addition to the principal symptoms, already mentioned, there was enlargement of the spleen and congestive hemorrhage from the intestines. In older children such conditions have not been observed. However, it is an important fact that at autopsies of older children we occasionally find deposits of circumscribed connective tissue which lead to *peritonitis at the porta hepatis*, and it is possible that by the formation of adhesions they may produce severe disturbances.

Whether the cases of icteroid cirrhosis without ascites may be due to an inflammation principally affecting the large biliary channels is not yet clear. Some authors designate their findings simply as *biliary cirrhosis*, but nothing precise as to the condition of the porta can be gained from the reports.

If we properly understand the anatomical basis of these occurrences the generally *unfavorable prognosis* is obvious. The appearance of the icterus is not the signal of the beginning of the disease itself but of the beginning of the end,—the obliteration of the biliary passages.

The only benefit of specific *treatment* is to aid the natural tendency of cicatricial contraction. For the restitution without functional disturbance, which can be attained only very early, the favorable time for such treatment is

usually past. This early period coincides, perhaps with rare exceptions, with fetal life. As a rule the end succeeds about three months' duration of the jaundice.

A *differentiation* of icteroid, syphilitic, hepatic cirrhosis from *congenital agenesis*, and from occlusion of the large biliary channels due to other causes, is only possible clinically when other evidences of syphilis are at hand or when, at least, it can be determined that the acholic discharges have appeared shortly after birth. Whether there is an accidental *complication of syphilis with one of the non-specific varieties of jaundice* must also be considered. However, in the latter case the liver would scarcely have a similar greatly increased consistence.

Hochsinger doubts the syphilitic basis of the jaundiced cases because of the continual absence of other simultaneous specific phenomena, but that they are not always absent is shown by one of my cases (icteroid cirrhosis with a papulo-syphilide). Attention must also be called to what has been mentioned in the description of portal cirrhosis.

Albumin and casts in the *urine* of syphilitic children is a very frequent happening, but it by no means justifies the conclusion that there is a specific affection of the kidneys. The same condition also occurs in non-syphilitics and in either instance is due to the action of complicating intestinal catarrhs and other ailments. According to numerous observations the urine of syphilitics who are otherwise well is always free of albumin. The interstitial changes which occur in the kidneys of children who survive (Hochsinger, Hecker, Schlossmann, Karvonen) are not susceptible of diagnosis. On the other hand, it is not unlikely that there is an *actual hemorrhagic nephritis* with blood, casts, and a large amount of albumin in the urine, and with dropsy, as in the well-known condition of adults. Here, as there, the timely coincidence with other manifestations of the disease, the peculiar richness of fat granules in the sediment, and the uninterrupted recovery after mercurial medication, favor such a view. This form of hemorrhagic nephritis has been described several times. Many of the cases, however, might be attributed to mercurial intoxication rather than to syphilis. Only a few reports of Bradley and Oedmanson, and a case of my own in which neither the mother, during her pregnancy, nor the child was treated, appear to be positive in this respect.

At several autopsies I have observed an extraordinarily extensive interstitial nephritis. It can be scarcely otherwise than that such cases undergo contraction in later life, and by careful clinical and anatomical observation we may expect to find syphilitically contracted kidneys during the first years more frequently than has been reported heretofore (Hellendal, Massolongho).

Occasionally a post-mortem examination shows that other organs have been attacked without causing any significant symptoms during life. Clinical interest is accordingly slight. *Pulmonary syphilis* is very rare in living children and the few cases soon succumb. The pneumonia which is so prevalent in syphilitics is probably always due to secondary infection.

The occasional infiltrations, vascular affections and ulcerations in the



*stomach and intestine* are not susceptible of a positive clinical diagnosis. The *enlargement of the spleen* is usually due to simple hyperplasia, exceptionally to a specific disease.

The literature contains few reports relative to changes in the *peritoneum*. It has been mentioned above that specific peritoneal deposits occur at the *porta hepatis*; another point of preference is the *capsule of the spleen*. Occasionally the path of the perisplenic induration may be directly followed by the elicitation of *friction sounds* and a *palpable parchment crepitation*. It must be remembered that severe derangements in the permeability of the intestine may sometimes be caused by adhesions.

### C. RELAPSE

In hereditary syphilis, just as in the acquired form *relapse* occurs in a certain number of cases which cannot be estimated with numerical exactness. Its appearance during the first period is frequently in the form of an *eruption*, and usually of a milder type than that of the primary attack. In addition, there is a recurrence of coryza, fissures, and onychia. *Condylomatous syphilis* and mucous patches (plaques muqueuses) also play an important, and ultimately a predominant or exclusive rôle, frequently combined with general or local *extensive swelling of the lymph glands*. The period of condylomata, and with this the danger of contagion, continues to about the fourth year of life. From that time the symptoms of *lues tarda* are to be expected.

If several relapses succeed one another a general abatement of their severity will usually be noted.

*Deep processes, particularly in the internal organs*, may occur in combination with this regular form, or exceptionally even alone. Apart from transitional forms, they no longer possess the character of diffuse infiltration, but instead they represent *gummata* or *syphilitic vascular disease*.

The *juxta-epiphyseal inflammation of the bones*, previously described, is again present, while the pathognomonic diaphysial periostitis of *lues tarda* remains absent. Exceptionally there are circumscribed *tophi of the cranium* or *tumescant gummata nodules* on the bones of the thorax and in the face. Disease of the bone marrow may occur as *circumscribed gummata of the spongiosa* or a *diffuse caseous osteomyelitis*, which I have noted also in the diploë of the skull.

Gummatous nodules, which occasionally ulcerate, have been observed in the *skin*. Of other organs only the *testicles* appear to be affected with any frequency, and during the first period of eruption are seldom involved to a demonstrable extent. Here either nodules are found which occasionally undergo ulceration and discharge externally, or there is diffuse nodular infiltration, sometimes accompanied by effusion into the tunica vaginalis. Quick and energetic treatment is necessary on account of connective tissue degeneration, which may cause a loss of function.

The condition of the *eyes* must also be considered. Here, besides iritis and parenchymatous keratitis, we find a focal *choroido-retinitis* of pathog-

nomonic importance. The views in regard to its frequency are divided. According to Silex it is rare; Hirschberg claims it to be common. Next to keratitis I have found it to be the most usual of all the late symptoms of hereditary syphilis. It begins in the fifth month and is indicated by squinting or holding of the head slant-wise. All other organic diseases must be regarded as rarities. *Gummata of the kidney, of the adrenals, of the intestines, of the liver, of the spleen, etc.*, have been reported. *Extensive vascular disease* may lead to *contracted kidney* (Massolongho). The same process was found by Berghing in the *heart* in combination with well-marked muscular degeneration; during the life of this 7-months-old child there had been attacks of cyanosis and dyspnea. Death occurred suddenly in the midst of apparent health. *Affections of the larynx* do not appear earlier than the second year.

Among the *diseases of the central nervous system* which occur as relapses there is gummatous meningitis, especially the basilar form; but the more common affection is endarteritis with its sequels (epilepsy, idiocy, cerebral hemiplegia and diplegia). Heubner observed *spasm of the glottis* of central origin.

Occasionally, in the earlier relapses, there is great *splenic* and *hepatic enlargement*, associated with marked anemia. Probably, instead of specific enlargements, these tumors are of the form known as *anæmia pseudo-leucæmia infantum*, which also corresponds with the blood findings.

#### D. TOXIC AND PARASYPHILITIC SYMPTOMS

The view from ancient times has been that the life of hereditarily syphilitic infants is endangered to a far greater extent than that of healthy children, a fact which finds its expression in the term "*polymortality*" of syphilitic progeny.

Two points must be distinguished.

The *principal factors of polymortality* are abortions, still-births, and deaths soon after birth. Common to all is the severe affection, fetal syphilis, from which the fatal outcome is not surprising. For example, in 153 pregnancies Le Pileur found abortions and still-births in 78.4 per cent.; 16.4 per cent. of the children died *post partum*, and only 5.2 per cent. survived.

However, a further factor had to be considered. It became obvious that even mildly syphilitic children and those who had never presented the symptoms of disease, but whose parents were infected, showed deficient development. The impression was conveyed that we were dealing with an unusual loss of resistance to pathologic influences which permitted an extraordinary acquirement of severe intestinal catarrh, pulmonary affections, and sepsis. *Sudden and unexplainable death* was believed to be remarkably frequent; but above all there was thought to be a conspicuous tendency to *severe anemic and particularly atrophic conditions*, which was more common in artificially nourished children than in those who were breast-fed, and thus led to the idea that artificial feeding was a serious menace to body and life.

*The polymortality was likewise manifested in this second group. Even*

those individuals who fortunately escaped were said to be feeble, poorly developed individuals in later life.

Therefore the conclusion was reached that a *syphilitic descendant shows a specific loss of resistance, a specific "debility,"* upon the basis of which deleterious factors that are readily overcome by those not subject to syphilis produce unusual effects.

These conspicuous phenomena were explained by an assumed *toxic "parasymphilitic" function* of the malady (Fournier): In consonance with this ingenious hypothesis the sperm or ovule, or both, may be "depraved" by the disease of the parents; they are subject to a *toxic, protoplasmic damage* which, although too slight to cause death, nevertheless exerts a "*dystrophic*" influence throughout the development of the embryo. This leads to deformities and retarded growth, general feebleness of cell function, probably also to a special debility of individual cell territories which, combined with slight alterations of other kinds, produces an acute or slow arrest of function. Therefore parasymphilis may be *hereditary*. It may, however, also *arise* in the hereditary florid disease as the consequence of toxins formed within the body, as in the acquired form, or both conditions may occur together.

This is the generally accepted law of parasymphilis, but many new facts have arisen which are calculated to shatter this view.

First, we must exclude from these considerations all cases which present florid symptoms at the outset. If in such children we find a general damage the other factors are of greater moment than where there is a parasymphilitic injury of protoplasm. Severe conditions are observed mostly in children with visceral syphilis, but here not so much the disease itself is the cause of the cachexia as the functional disturbance of vital organs. Even breast-fed children cannot always be saved. There are cases, however, in which energetic treatment clears up the condition and henceforth there is general improvement and development.

In the ordinary form of infantile syphilis, in relatively severe cases, there appears to be a certain arrest of weight during the eruptive period, as in adults, which may be due to the general damage; but uncomplicated cases, therefore those which are entirely free of criticism, are difficult to find. For the milder secondary phenomena, judging by numerous observations of my own, such an inhibitive influence does not exist. The increase in weight may proceed uninterruptedly. I do not hold the prevalent opinion that in atrophic children the simultaneous eruptions and atrophy disappear under mercurial treatment, provided, however, we are not dealing with the aforesaid cachexia of visceral syphilis.

In regard to actual parasymphilis, irrespective of obvious products of the disease, it must be admitted that in some cases *we cannot arrive at a conclusion without assuming an actual decrease of resistance*. Here, however, as Finger has recently made clear, it is difficult to separate the condition dependent upon syphilis from that due to other concurrent predispositions which frequently act in the same manner (alcoholism, lead intoxication, tuberculosis,



neuropathy). A point of importance is this, that a like inferiority occasionally appears in families not so predisposed, and again that in many syphilitics it is absent.

However, *for most of the conditions, and particularly for those which prevail in nurslings, the assumption of such a specific weakness* may be dispensed with. Sudden deaths and severe anemic conditions are common in non-syphilitics. The increased predisposition to disease and in particular the tendency to atrophy remain to be explained.

The belief in these is based largely upon the observations of hospital physicians and principally the older ones, to whom the excessive mortality of syphilitics appeared to require additional explanation. It is known, however, and in the last few years has been the subject of particular discussion, that non-syphilitic infants also show an extraordinarily high mortality in institutions. If the conditions are so far improved for the latter that a decreased mortality results, the syphilitic children likewise participate in the improvement. I have observed such changes myself and now meet with the same successes in the hospital with syphilitic children as with others. Naturally, this is also true in private practice. I entirely agree with Freund's statement that under similar conditions the outlook for the syphilitic and the non-syphilitic scarcely differs. I say "scarcely" with intention, for some reserve is necessary. There is undoubtedly a predisposition to purulent infection and sepsis, which produces an increased mortality. The greater frequency alone would not explain a specific loss of resistance; in syphilis it might be accounted for by the more numerous ports of entrance (erosions, fissures, etc.). It is true, the sepsis of syphilitics runs a severe course, so that the idea of a decreased resistance is hard to reject. Almost all of my fulminant cases, especially when associated with hemolysis, occurred in syphilitics.

Particular emphasis must be given to the fact that *better results in the raising of syphilitic children* are also obtained from *artificial* nourishment. The number of authorities who more or less strongly uphold the utility of this method is increasing. Even Fournier, the author of the term "parasyphilis," has lately been inclined to admit that in this form the advance in knowledge appears to have brought about changed conditions.

The conclusion, therefore, is this: "*In so far as the actual polymortality exists it is due, in addition to abortions and still-births, to the "projection" of visceral syphilis in infancy.* Children who are free of this show no essential difference from the non-syphilitics in their vitality apart from an increased disposition to septic diseases.

#### E. MIXED INFECTION

The uncommonly rich symptom-complex of this disease is still further diversified by the previously mentioned predisposition to septic infection which, either local or general, may occur with remarkable frequency in all of its forms, preferably during the early eruptive period. The many ports of entrance in the skin and mucous membrane, and, not least in importance, through the

nose, give uncommon facility for its development. Of the several forms only two will be mentioned: First, *purulent synovitis*, which is often associated with true syphilis of the bones and complicates its clinical picture. This probably is never of a specific nature but is a secondary invasion to be ascribed principally to streptococci.

Second, *hemorrhagic syphilis* of infants must be mentioned. Hemorrhages which closely resemble the hemorrhagic diathesis are particularly common in the syphilitic nursing. The hemorrhage increases from petechiæ to large and uncontrollable effusions into the skin, the outer and inner surfaces of which present all the gradations of bleeding. Death may occur by hemorrhage from an umbilical wound. Behrend has believed it necessary to distinguish a special form of syphilis by these symptoms and many opinions have been expressed for and against his view.

Some authors (Schütz, Mraček, more recently Esser) assign the cause of hemorrhage to a disease of the vascular walls without, however, explaining that local hemorrhage may occur in this way but never a general hemorrhagic diathesis with decreased coagulability of the blood.

Other authors (Fischl) doubt the importance of vascular changes, and the majority of them believe that a complication with some other cause of hemorrhage is much more likely. Upon the basis of bacteriologic investigation in six of my own cases, the result of which is well supported by the literature, I believe that, in so far as we are not concerned with stasis hemorrhage in hepatic cirrhosis, this other cause, in most or perhaps in all of the cases, originates from a secondary *septic infection*, which, for unknown reasons, in syphilitics readily leads to hematolysis. Accordingly, an actual, severe syphilis hemorrhagica does not exist, at least in nurslings. Whether relatively insignificant petechial hemorrhages or hemorrhagic exanthems are sometimes produced by the specific visceral changes, to which Mraček alludes cannot at present be determined.

#### F. DIAGNOSIS

No special explanation is required of the importance and under some circumstances of the serious consequences, not only to the child but to the relatives, and also to the physician for other than therapeutic reasons, which attach to the recognition or non-recognition of a disease of such virulent infection and of such great influence upon family relationship. From the foregoing we have learned what anamnestic and clinical data, from the period of incubation until the symptoms are fully developed, are required for a diagnosis, or at least a well-founded assumption. But even in the days which are free of symptoms—in the interval between the relapses—there are a number of changes, individually ambiguous or meaningless, which may be somewhat suspicious when they are combined, even without a knowledge of the history.

These, in the order of their importance, are *anemia*, especially with a yellowish hue, *splenic tumor*, greatly enlarged *cubital glands*, *alopecia*, the *downward glance* resembling hydrocephalus, the more or less accentuated *saddle-nose*, *contraction of the nasal passages*, and *radiating cicatrices* which extend

from the red lips into the neighboring skin. The traces of earlier fissures are especially significant at this age, when other causes of cheilitis are rare. The consideration of these symptoms will furnish a correct explanation of many of the late localizations of the disease and prevent the serious error of employing a wet-nurse.

It is well, perhaps, to mention some of the conditions which have frequently led to an erroneous assumption of syphilis. Every hindrance of nasal respiration and every rhinitis in early life cannot be attributed to syphilis. In many healthy newborn infants and those of premature birth there is a *certain degree of dry swelling which causes snuffing* and probably is the consequence of congestive and desquamative conditions. *Congenital adenoid proliferations* may also cause error. The secretion of *simple acute coryza* is more profuse, thinner, and non-hemorrhagic, and is usually associated with catarrhal changes in the pharynx. *Septic, diphtheritic, and gonorrheal rhinitis* must also be considered.

The artificial ulcers of *Bednar's aphthæ*, and the thickening of the *lingua geographica*, which is so common, are often mistaken for syphilitic plaques. An *ulcer on the heel* occasionally is given the same importance, and the *ulcers of ecthyma* are sometimes regarded as ulcerating syphilides because of their similar shape.

*The glistening redness of the soles of atrophic children* is very often ascribed to syphilis, but the diffuse plantar infiltration and typical coarseness and desquamation of syphilis are absent. More comprehensible is the inclusion of some peculiar *forms of intertriginous eczema* which are gradually distributed from the boundaries of the diffuse macerating surface as papulo-squamous, sometimes as eroded efflorescences varying from the size of a pin-head to that of a lentil. These closely simulate syphilitic eruptions and may by confluence become annular and scaly, and in cachectic individuals produce superficial ulcerating infiltrations which resemble serpiginous exanthems. The absence of other stigmata, the limitation to the vicinity of actual intertriginous areas, the exemption of the palmar and plantar surfaces, as well as the ineffectiveness of specific treatment, make the differentiation not altogether difficult.

### G. PROGNOSIS

In a prognostic respect we are least interested in the outcome of the disease. That question can be answered briefly. The ordinary forms heal rapidly and completely even when there is involvement of bone. For the visceral forms the verdict must be the same as in homogeneous affections of later life. In how far the *question of nutrition* modifies these views has already been touched upon.

Much more essential are data which will sustain a general opinion as to the later progress of the case. *Will a relapse occur, and when is it to be expected? What likelihood is there of a syphilis tarda? Does the congenital disease notably diminish the prospect of life? Can a healthy and normal human being eventually develop from the little diseased patient?*



Very little knowledge is available for the decision of these questions. Investigations which must necessarily be continued for many years have as yet been made by few physicians (Hochsinger, Karcher, Pott). We owe the most valuable researches to Hochsinger, who followed the history of sixty-three individuals for four to twenty years. Therefore, there is still a hiatus which might be filled by experienced family physicians, who certainly could give valuable information on the subject.

It is a conspicuous fact that the generally unfavorable reports which relate to the future of infants, and also of older children, are in no wise substantiated by actual close observation.

*Relapses* apparently do not form the rule, and are probably less likely the milder the case. Hochsinger reported relapse in about one-third of his cases. In view of this comparative rarity it is interesting to note that among the inmates of hospitals there are fewer relapses than there are first eruptions. In 85 cases of syphilis which occurred during the first three months of life I observed only 20 relapses between the sixth and fifteenth months. Naturally, the death of many young infants must be included in this reckoning.

One relapse only may occur; on the other hand very intense symptoms, especially condylomata, may constantly recur. After the first period the free interval is rarely less than three months and may be twice as long. After the first year, during which 70 per cent. of all relapses occur, the probability of new eruptions is slight.

According to unanimous reports *lues tarda* seldom appears after the fourth year,—the end of the condylomatous period. Of Hochsinger's 63 cases only 10 were affected after that period and the author believes these had been treated late or insufficiently.

Therefore *congenital syphilis is undoubtedly curable*. The children may become healthy and even robust. The number of weak, anemic individuals is no greater among syphilitics than others. Whether among syphilitics there is a general infantilism<sup>1</sup> or only the organs of generation are affected; whether protracted puberty and other inhibitions of development are more frequent and in causal relation, is uncertain. It is doubtful whether there is a special predisposition to tuberculosis, as some investigators accept

### III. LATE SYMPTOMS OF HEREDITARY SYPHILIS (LUES TARDA)

As previously mentioned, in children who are hereditarily syphilitic new symptoms occasionally appear after the period of relapse—usually from the fifth year—which, in their nature and course, show an astonishing uniformity with the tertiary products of the acquired disease.

These late forms cannot be conclusively designated as *syphilis hereditaria tarda*, as that condition is defined quite differently by a number of experienced observers. According to these authors syphilis tarda does not include the late

<sup>1</sup> Here, naturally, we do not consider individuals with cicatrized atrophied testicles.

relapses, but runs a peculiar course, the first manifestations of which generally are the tertiary phenomena, while during the suckling period no symptoms are present. The actual existence of this *tertiarisme d'emblée* with its postulate of a surprisingly long latency—an absence of secondary symptoms—is still a debatable subject, and so far it has not been possible to overcome all the objections to such a view. It is true, the eruptive phenomena are often so slight in the nursling as to be readily overlooked. In fact exanthems are often absent. In one case which I observed from birth to the fourteenth week the child, a hereditary suspect, snuffled very little and showed only a slight infiltration of the lip, but the finding of an osteochondritis at the necropsy confirmed the diagnosis. Death was due to an intercurrent pneumonia. The possibility of an intra-uterine termination of the secondary symptoms must be considered. Even an early acquired infection, of which there was no knowledge, may in some cases be the underlying cause.

The majority of the *late symptoms of hereditary syphilis* do not demand a detailed description. They resemble the corresponding tertiary phenomena of the acquired disease. *Periostitis* in its several forms—rarefying, ossifying, gummatous—is especially frequent and well-marked, and affects principally the tibia, but the lower and upper arm are not exempt. Chronic *synovitis* and *arthritic* disease are observed under the picture of tumor albus and their bilateral occurrence raises the suspicion of a syphilitic foundation. Further, we find *gummatous cutaneous and subcutaneous syphilides*, and gummatous affections of the *tongue*, and of the *mucous membrane of the nose and pharynx*, associated with necrosis of bone and subsequent perforation and radiating cicatrization. Profound *affections of the larynx* and their product, cicatricial stenosis of the air passages, are important and less rare conditions. Characteristic changes of other organs occasionally occur.

*Disease of the internal organs* requires more comprehensive study. It appears that the *liver* is affected relatively often, as well in the form of tumor-like gumma, which gives rise to lobulated liver, as that of the hyperplastic stage of cirrhosis. Many familiar bilious icteroid cirrhoses have also, with some justification, been placed in this category. Amyloid disease must also be considered. Enlargement of the liver is probably always associated with some degree of *hard and enlarged spleen*. Confusion with sarcoma of the liver, with non-specific cirrhosis, and with anemia splenica is very easy.

Among *renal diseases* certain forms of infantile *contracted kidney* are probably of syphilitic origin, but in this realm little is definitely known.

Special importance is attached to late infantile syphilis because of the damage to the *brain*. The number of observations concerning *chronic meningitis and endarteritis* has recently increased decidedly. Especially do we know that even at this early age the so-called "*infantile*" *tubes*, that is, the symptom-complex and the *progressive paralysis* which is closely allied to it, develops in no wise rare cases upon a hereditary syphilitic basis (Alzheimer, Thiry, Kalischer, Brasch, v. Didynski, and others). In a small percentage of cases a connection with *epilepsy* can scarcely be denied (Fischl, Bratz and Lüth, etc.).

For the *diagnosis* of the specific nature of such changes, which are not obviously syphilitic, we are in possession of but little distinctive evidence in addition to the history. *Cicatrices, radiating from the red of the lips*, or cicatricial bands are significant, but, since profound cheilitis occurs in scrofula and in the course of severe infectious diseases, their importance is decidedly lessened.

Much discussion has been devoted to the diagnostic value of *Hutchinson's triad*: *Keratitis interstitialis, central deafness, and a peculiar deformity of the teeth*.

A résumé of the numerous reports of experienced physicians would probably show that *keratitis interstitialis*, the most prominent link of the triad, although not constantly of syphilitic origin, is nevertheless so in a vast majority of cases and is of great diagnostic significance. Next to keratitis in importance is a certain form of *choroiditis areolaris* characterized by multiple small foci. *Deafness* is much less common and is not in itself exclusively a syphilitic finding (Schwabach). The importance of *tooth deformities* is diminished because they possess no complete pathognomonic individuality. In hereditary syphilis there are various disturbances of growth and other changes in the teeth, but on the other hand analogous conditions may be produced by nutritive derangements. Only one form is to be regarded as at all characteristic; here the *permanent* teeth, particularly the central incisors of the upper jaw, show usure or a facet which is more marked posteriorly than anteriorly and causes a notched or semilunar cutting edge. Such a phenomenon is, however, rare.

#### IV. TREATMENT

**Prophylaxis.**—In so far as possible the physician should endeavor to prevent the procreation of children who in all probability will be syphilitics.

*When may syphilitics marry without endangering wife and children?* As a rule there is no reason to prevent the marriage of males who have been thoroughly treated, whose infection antedates at least four years, and who have had no relapse for two years. A renewal of the treatment shortly before marriage is advisable. Frequent relapses prolong this period accordingly. In adhering to this minimal period, *even without being called upon directly to decide the question*, we must not permit ourselves to err by the knowledge that ill-advised or wilful patients occasionally rear healthy children even when they have been recently infected.

If *married couples* consult the physician on account of abortions, stillbirths, or illness of their children, energetic treatment of both parents, or, if the mother has not been infected, only the husband, must at once be instituted. In a relatively high percentage of the cases the result will be the birth of healthy children. How the physician will escape the cliffs of the oftentimes awkward situation and reach his goal is a matter for his diplomacy.

If the mother is *already pregnant* she should be treated at once. This is all the more necessary if florid symptoms are present at the time, but treatment must be instituted even when their occurrence can only be surmised by the history. There is no uncertainty except in the cases of pure paternal heredity,



and here also Fournier advises treatment. The form of internal medication practised by that author throughout the period of pregnancy<sup>1</sup> is not generally accepted in countries outside of France. While in older cases the ordinary form of treatment is sufficient, experience has unanimously decreed that a mother who has recently become syphilitic has little hope of bearing a healthy child. In such individuals Riehl's process<sup>2</sup>—a combination of general treatment with a local application of vaginal globuli—appears to furnish excellent results as regards vitality and morbidity.

**Treatment.**—In the treatment of children the internal administration of mercury and the corrosive sublimate bath are of primary importance, in contrast to the treatment of later life. I prefer the former, as it can be readily carried out and is very effectual. The dose per day is 0.01-0.02, administered in small amounts and never upon an empty stomach.

Of the numerous preparations<sup>3</sup> I have employed only hydrargyrum iodid flavum (mercury protoiodid), calomel, hydrarg. oxid. tannic, and salicyl., which show no noteworthy differences in their action and subsequent effect. The protoiodid appears to be particularly energetic in disease of the bones.

The *corrosive sublimate bath*,<sup>4</sup> an especial feature of which is its prolongation (ten to fifteen minutes), appears to be of greatest value in extensive external eruptions or when there is decided loss of epidermis (intertrigo and the like); if such conditions are absent this treatment is probably too slow and should be used only as an auxiliary measure.

All other methods of application are generally regarded as only exceptionally of value in the child. Like many other pediatricists, after a brief trial we have abandoned the treatment by inunction (0.25-0.5 per day) as it irritates the skin too greatly. This treatment is recommended for fat and very robust children, and after the first six months if there be relapses. It may be tried in severe cases where a quick effect must be obtained. *Packing with mercurial plaster-mull* (Unna),<sup>5</sup> a substitute for inunction treatment, often produces an eczema.

*Treatment by injection* is inexpedient because of the relatively thin soft parts and the possibility of painful infiltrations.

Siefert's modification of *Welder's sack method*,<sup>6</sup> and Blaschko's *mercolint bandage* are suitable occasionally in mild cases.

As a rule the administration of *mercury is well borne* by infants. The

<sup>1</sup> Alternate treatment with protoiodid of mercury 0.25 per day and iodid of potassium 1-2.0 per day.

<sup>2</sup> *Wiener klin. Wochenschr.*, 1901, No. 26. Vaginal globules (Ung. cin., 1.0 Butyr. cacao 1.0-2.0 daily throughout pregnancy), inserted to the portio and fixed with a tampon.

<sup>3</sup> Compare the compilation of Monti, *Kinderheilkunde in Einzeldarstellungen*, II Bd.

<sup>4</sup> 0.75-1.0 to the bath. Wooden or enameled tub! Careful handling of the drug is demanded.

<sup>5</sup> The four extremities are enveloped in turn and covered with a bandage. The applications should be renewed every four to eight days.

<sup>6</sup> *Naturforscherversammlung*, Sect. f. Dermatol., etc. Munich, 1899. Six to 10 gm. Hydrarg. concreta upon the woolly side of a piece of lint (20 x 40), folded and sewed into a bag, and tied on. Renew every four to six days.

danger of *stomatitis* and even of marked *salivation* is so slight that special care of the mouth is scarcely required. In inunction treatment Monti warns against the possibility of sudden death from cardiac weakness and edema of the lungs, but these are probably accidental complications rather than the effect of treatment. Also the gradually developing *anemia* can hardly be ascribed to the metallic intoxication, as the same symptom is characteristic of syphilis (Schiff) or is due to the intestinal disturbance so frequently associated with the disease in infancy. In any event I have never found reason from this to interrupt specific treatment and administer iodid of iron simultaneously, as advised by Monti. Widerhofer's method, however, of combining iron with mercury (Ferr. carbon. sacchar., in doses of 0.1-0.2) is quite proper.

Internal treatment, and particularly the protoiodid, is said to evoke *intestinal irritation*. I have never been able to convince myself of the correctness of this statement, but do not deny the possibility of such an occurrence. Despite mercury we very often succeed in improving the condition of the intestines by dietetic means. Calomel in small doses is in good repute as an intestinal antiseptic in nurslings. For this purpose I employ calomel in combination with tannigen (2.4 — 0.005) and have seen excellent results. Some authorities add a small quantity of opium.

The internal administration of mercury *prohibits the simultaneous employment of salts of iodine and bromine*, and *vice versa*, because of the danger of corroding ulcerations of the gastrointestinal canal. Also, the child should never have mercury when the nursing mother is taking iodine.

*Treatment with iodine* (0.2-0.5 daily) is of little service in the ordinary syphilis of infancy. It is to be considered in those forms which, like many of the relapses, resemble the gummatous stage of later life, and is especially useful in the rare cases of *syphilis of the brain*. Under such circumstances a *mixed* treatment in the most serviceable and in the severe cases inunctions are preferred. In young children prolonged iodine medication may greatly interrupt the gain in weight.

Important *local affections* are treated in the same manner as in the adult: covering marked infiltrations (onychia, etc.) with gray plaster, dusting the condylomata with calomel after cleansing with normal salt solution, etc. The fissures of the mouth are treated advantageously with a 10 per cent. solution of chromic acid, the surrounding skin being carefully avoided, or with a one per cent. corrosive sublimate solution. In the nose mercurial salves may be applied, after softening the crusts. The nasal affection is exceedingly tenacious. Here great benefit has resulted from painting with solutions of silver nitrate (0.5-15), as advised by Henoch, and in the clinic we have utilized this remedy successfully by spraying<sup>1</sup> with a solution of 1 to 1,000.

The *influence of specific treatment* becomes noticeable in a few days. The exanthem fades, new eruptions do not appear; fissures and mucous plaques are

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<sup>1</sup> The child is held by the nurse with its head over a basin, so that secretions and fluid may flow through the mouth or nose.

distinctly improved after a mercurial treatment of four or five days; in the case of pseudo-paralysis movement returns with surprising rapidity. Rhinitis, as already mentioned, is much more tenacious, and it is not unusual for a snuffling respiration and a nasal timbre to remain for some months after the secretion has been arrested, perhaps as the expression of a chronic, non-specific proliferation of the mucosa or of the pharyngeal tonsil, or of permanent stenotic change in the bone. The visceral forms appear to require a comparatively long treatment before improvement is noted.

This leads to a few words as to the *length of the treatment*. In general it has been found advisable to continue treatment for two weeks after all the symptoms have entirely disappeared. This usually requires five or six weeks. Nevertheless, according to my experience it is doubtful whether this period is sufficient for profound changes which may have occurred. For example, in a child who perished from an intercurrent disease a fortnight after a six weeks' treatment with protoiodid of mercury and four weeks after the disappearance of all exanthematous symptoms, I found a decided osteochondritis. As the certainty of the absence of important visceral processes is never absolute, even when symptoms no longer exist, prolonged treatment is advisable, of course within the limits of intoxication.

Whether, in the absence of a relapse, *renewed treatment* should be instituted in the same manner as the intermittent treatment of adults, and whether by this means relapses are more apt to be avoided, cannot be asserted positively. Most authors favor a second treatment after the first year, and again in the second and third years. Monti believes that the administration of *iodid of iron*,<sup>1</sup> continued until the disappearance of the anemia and splenic tumor, has a preventive effect.

**Nourishment.**—The nourishment of hereditarily syphilitic patients presents certain peculiarities which are of salient practical importance and must therefore be explicitly described.

Most text-books and many physicians *oppose artificial nutrition*. They claim that this is dangerous to life as the constitutional weakness of the child has already enhanced the possibility of failure. Upon the other hand the threatened transmission precludes the employment of a wet-nurse in case the mother cannot nourish her child, and even the mother is not always immune from infection. In the face of such a dilemma the active discussion regarding the principles which are to govern the decision of the physician can readily be understood.

*If the mother can nourish her child* the decision is simple. According to Colles's law, when the mother has never manifested symptoms she is usually exempt from contagion. Nevertheless *a few exceptions are known*: Primiparæ, in whose children a late appearance of the symptoms shows them to be mild, have been infected through nursing. It is easily understood that in such

<sup>1</sup> 0.1-0.2 once to three times daily according to age. It must be remembered that, according to many syphilographers, iodine alone has no protective power against relapses, this being the property of mercury alone.



cases the immunization of the mother has not been sufficiently long and has been maintained by a poison only slightly active; therefore, sufficient protection has not been conferred. This possibility is not considered by most authors in practice, and is so rare that a man of Fournier's experience has not observed a case. Hochsinger alone is absolutely opposed to a healthy primipara nursing her sick child.

Contrary to Profeta's law, contagion has sometimes happened to the child. Because of its rarity the removal of the infant from the breast is not necessary. The danger of infection from other contact is much greater and its prevention is more important than a prohibition of nursing. In late post-conceptional syphilis of the mother Czerny and Keller advise that she should not nurse the child.

*What shall be the procedure when the mother cannot nourish her child?*

On account of the great danger of artificial feeding many authorities advise a *wet-nurse*. They reckon upon the comparative rarity of infection (Henoch, Widerhofer), or recommend that the decision be left to the wet-nurse after the condition has been explained to her.

Anyone who has the temerity to expose a healthy person to infection, to risk infection not only of the nurse but others, and hereditarily of an entire family, he who desires to escape responsibility by leaving the decision to one incapable of forming an opinion, or who is of the naïve belief that after disclosing the state of affairs to the family the necessary discretion of the latter can be maintained, may follow this advice. I share in the general opinion that under no circumstances is such a method justified and that artificial nutrition is the only recourse. This decision should not be difficult, because, as has already been stated, with a proper method of nourishment there is no more danger to the syphilitic child than to the non-syphilitic.

Therefore, no wet-nurse—not only when we are dealing with manifest syphilis, but even when the child shows no evidence of disease at birth but whose family history is known. When infection occurred many years previously perhaps an exception might be made. If the eruption appears *during the period of breast-feeding the nursing must be stopped at once*, as soon as the first symptoms appear; we must not wait until a typical exanthem is present. The opportunity for withdrawal of breast nourishment may perhaps be brought about by difficulty in suckling due to coryza and fissures.

There will always remain a proportion of syphilitic children—premature births and cases of severe visceral syphilis—whose improvement is essentially more hopeful when they are nourished with human milk; milder cases thrive with the bottle. With natural nourishment only may we hope to maintain them until a therapeutic influence is exerted. In such cases a means must be found of administering milk that is drawn from the breast.

The symptoms of *lues tarda* are favorably influenced by iodine (1-2.0 daily). Progressive and visceral forms require a mixed treatment which may be conducted advantageously in the regions of iodine springs (Tölz, Heilbronn, Hall).

[The treatment of pregnant syphilitic women must be persistent; particu-

larly of those who have aborted previously. Even though they cannot be seen and watched regularly, no harm can be done. Salivation can be avoided. It is my habit to give them an iodid and the yellow iodid of mercury in alternating weeks for many months in succession, for instance the first, third, fifth, fifteenth, etc., week or 15 or 20 drops of the potassium, sodium or strontium iodid saturated solution 2 hours after a meal, 3 times a day; the second, fourth, sixth, sixteenth week 1 or  $1\frac{1}{2}$  grains of the protoiodid of mercury daily, in three pills, to be taken in the same way. I generally add a small dose of an opiate or of atropin to each pill.

The facility and safety of mercury when the patient is a newly born or small child, salivation (gastritis or enteritis being almost unheard of), facilitate and advise protracted treatment. I am never satisfied with the disappearance of manifest symptoms of syphilis, but continue treatment for several months uninterruptedly, then intermit a month, and begin again in alternating months. After a while it is safe to mediate a month out of three. Unfortunate experience has prevailed upon me to give mercury several months in the second and in the third year.—EDITOR.]

## SPEECH DISTURBANCES OF CHILDHOOD

By H. GUTZMANN, BERLIN

THE child attains the faculty of speech a long time after birth, slowly, just as it learns the movements of walking, running, grasping, and holding. Speech itself is only movement, although quite complicated and highly coördinated. The three great musculatures of *respiration, voice and articulation* must be induced to a common activity; the gradations of muscular movement of the voice and of articulation are so fine that with a relatively slight muscle sense in these parts the art of speech will be accomplished but slowly and laboriously. The newborn child is mute, and since in the first days a great number of children at least seem to hear but little or not at all, we have not alone a *physiologic mutism* but a *physiologic deafmutism* of the newborn. How the senses gradually develop, in what manner the faculty of perception arises, how, upon the basis of these the conceptions become manifold, and are profoundly and more intimately associated, and how, finally, the function of volition springs from this psychic structure, furnishes a great and interesting realm in the *psychology of the child*. In the works of Steinthal, Lazarus, Preyer, Goldammer, Vierordt and, last not least, Kussmaul, will be found a great number of individual observations and careful descriptions of this psychologic order of development. When we concern ourselves more minutely with these questions we shall find that almost all errors of articulation of children may be referred to certain physiologic disturbances in the development of speech. I say physiologic disturbances because they must necessarily arise with development; and, in order to completely appreciate their value and influence upon the further speech activity, a brief consideration of these processes is necessary. This will at the same time serve as an introduction for a sensible and complete *prophylaxis of speech disturbances of childhood*, and here it may be stated that almost all speech disturbances considered in this article can be prevented by this prophylaxis.

The development of speech may be divided into *four* distinct parts, although as to time they are not so separated that one period begins as another closes. The first division is the *crying period*. Even the ancient philosophers have debated the causes of crying in children and have offered the most complicated theories in explanation. Thus, children were said to cry because they felt in advance the misery of the world that awaited them; they cried in protest at coming into such a world; and it was even held that the cry of the boys was "oh ah," the girls more like "oh eh." As the newborn child naturally



could not be expected to speak a fluent Latin this was interpreted to mean "o Adam cur peccavisti!" and "o Eva cur peccavisti!" The crying of an infant is certainly an indication that it is *dissatisfied*. The *negative sense of being* completely dominates it. In its relation to speech this crying epoch has special importance, as the respiration of crying is preliminary to later speech respiration. The infant cries in prolonged tones, which are frequently interrupted by a short intake of breath. These inspirations are through the mouth, while in sleep and when it is otherwise quiet, the child breathes through the nose. The ratio of inspiration and expiration of crying is that the former is short and through the mouth, the latter quite prolonged but also through the mouth. *This is likewise the type of the later speech respiration.* We are therefore justified in regarding the respiration of crying as preliminary practise for speech respiration. The longer the expiration during crying, the better for the later speech; if the expiration is prolonged, many words can be uttered during a single breath, and the better we can control the breath the more fluent will be our speech. Practise of the voice, which is a feature of crying, is by no means purposeless; the strengthening of the voice which thus results, the delicate tone which is heard in the softest expiration, are preliminary exercises of great importance for the later speech. We shall not discuss the influence of crying upon the general development since that is beyond the confines of our subject.

When the child has become more tranquil and begins to take an interest in its surroundings it often lies awake without crying and attempts, besides movements of the arms and legs, also to employ its organs of speech. Quite unconsciously the first developed sounds bear a very uncertain character and are produced in all portions of articulation, although with a preference for the lip and the anterior tongue sounds. Apparently these are muscles which the child has used earliest. Sucking is a natural function, for the movements which produce it are noted even before birth if the finger is accidentally introduced into the mouth of the child during obstetrical examination. Among the vowels the simplest is *a*, which requires no other movement than the opening of the mouth. This vowel, or at least its modification *ä*, is most frequently combined with the consonants *b, p, m, d, t, n*, so that the sequence of syllables such as *ba-ba-ba, da-da-da, ma-ma-ma*, etc., sound like *dä, da, nä, na*, etc., which is the language of children at the age of three or four months in their afore-said quiet condition. Besides these simple utterances we hear, as already stated, other articulations, a series of peculiar gurgling and sputtering sounds, also sounds that first occur voluntarily much later; for instance, the palatal *r*. The child is sometimes so pleased with this sound production that it crows.

These articulations are nothing more than congenital instinctive movements, or, if we desire to maintain the conception that a movement without preceding stimulation is impossible, they are reactions or reflexes following some endogenous irritation. The child and those about it are pleased with them, and from the most ancient times the maternal love has attached a special meaning to this first childish lalling, in that the mother attempts to

recognize her name in these disconnected, groping, ataxic beginnings of speech. Thus it occurs that the first childish efforts at speech, which are mostly automatic or reflex, very soon attain the importance that those around the child attach to them. The syllabic sequences ma-ma-ma, ba-ba-ba were declared to be the names of the mother and father, a meaning which the child had not yet attached to these sounds. We must not, therefore, consider it remarkable that the names given by children to their parents are the same or very similar in almost all languages, not alone of civilized peoples but also of the aborigines. Papa and mama are not only the names of endearment in English, German, Russian, and French, but also in the language of the natives of Central Brazil, of the "Bakairi" and "Trumai," in the speech of the negroes of Africa, of the South Sea Islanders, and elsewhere. Often, instead of papa, it is baba or bawa, instead of mama it is papa, meme or eme, also mene and similar sounds, but always *the names of the father and mother are a sequence of sounds which correspond to the first lalling of the child*. It will thus be observed that the child creates its own language, and often when we investigate and ponder over the origin of words, an expressive answer would be given by pointing to a child who is learning to speak. In the speech development lies the simplest solution. Kussmaul describes this epoch in the linguistic development of the child as the *period of original or natural sounds*. Perhaps a better designation would be the *period of reflex speech*.

Meanwhile the *mental faculties* of the infant have markedly developed. The organs of special sense, which at first only apperceive, now begin to appreciate: sight has generated observation, listening is developed from hearing, touch produces sensation, and eating creates taste. Thus the organs of sense are daily unfolding their object: to construct the complete psychological world of the child and to bring the individual perceptions into relation. The great realm of the association of the individual expression of the senses builds itself in sequence. The child now not only hears but listens, that is, hears *attentively*, it now not only sees but observes, therefore, sees *attentively*, so that a very remarkable phenomenon—imitation—arises which may be regarded as a kind of higher reflex. In a stimulation of the sensory impression is also found the impulse which produces the reflex, that is, the cause of the movement.

What appears in imitation as such a remarkable power we can distinguish better and more simply as the force under which a reflex movement appears through stimulative impressions. The child sees movements and imitates them; it hears sounds and at once attempts to repeat them in so far as its skill and capability of coördination of the organs of speech permit. In the growing child the desire to imitate bodily infirmities, such as squinting and limping, is very common. It is also well known that general nervous spastic phenomena, for example, chorea, may become epidemic in schools.

In some individuals the reflex movements respond to stimulation rapidly, in others more slowly. This is also true of the power of mimicry which in some children responds early and energetically to sensory impressions, while in others it is retarded and awkward. To comprehend this peculiar



variation we must remember that children, even relatively early, are able to understand a great many words before they can voluntarily pronounce them. It will not be without interest to investigate this faculty of word perception in a child about 10 months. When it is quiet and attentive its comprehension of words can readily be tested, as a child will look toward an object usually when it recognizes the word which designates it. Naturally apperception is necessary in this test; mere perception is not sufficient, as the child varies greatly in its attention. Thus knowledge and understanding of a word will be demonstrated by a turning of the eyes toward the object mentioned which at another time may not occur. If this experiment is continued patiently and with perseverance, and is done as if in play, without tiring the child, a clear insight of its sensory accumulation of words will be obtained; we will discover which sounds have already made so profound an impression that their repetition at once awakens the memory of something known. In the normal child this relatively early cultivation of the sensory speech center is never accompanied by a corresponding development of the center for motor speech. It appears almost as if the stacking of word sounds propagates the endogenous stimulation for the development of the motor center. However, the child must eventually endeavor voluntarily to imitate what it has perceived in its surroundings as regards speech production, and the many defects in these early, groping, ataxic efforts cannot appear especially remarkable, since the other movements, such as grasping and walking, are at first extraordinarily ataxic and only develop into coördinated movements after much exercise and many failures.

This *period of imitation*, the third therefore in the evolution of speech, is probably the most important in the entire development; for now, if at all, there is opportunity for the child to acquire errors, or to absorb the imperceptible germ of faulty speech development. This imitation must follow a model. If the model is poor, if it is in any way abnormal, we must not be surprised when we find anomalies in the subsequent speech of the child. The better the example, therefore the more distinct the articulation of those who are about the child, the clearer their syllables, and the more beautiful their language, the more certain may we be that the little mimic will acquire good speech.

It will therefore be recognized that the movement toward a *natural prophylaxis of speech disturbance* must begin in this period of imitation. The environment of the child offers the first impetus for the acquirement of speech defects, regardless of hereditary predisposition, and the like, which will be considered later.

The second exceedingly important factor in the development of speech defects is the disproportion, which we have learned to recognize, between the growth of the motor and of the sensory centers. If from the well-developed sensory center continuous waves of stimulation arise which endeavor to excite the motor tract of speech, and if, on the other hand, the proficiency of the organs of articulation, the coördination, has not yet been perfected to such an extent as to translate this stimulation to corresponding movements, we must



not be surprised if errors of the most varied nature arise in the speech development. The child often makes mistakes in speaking; perhaps it uses the wrong term; or if the word is improperly produced it repeats the initial syllable or sound, at first without special stimulation, but later with unquestioned acceleration of the speech movements. This is primarily a purely physiologic peculiarity which has been closely observed by all of the aforementioned investigators of infantile speech. Preyer has quite properly placed these faulty articulations in parallelism with the speech disturbances of adults. As the phenomenon is at first physiologic it is to be observed in almost all children. I have rarely missed this halting early speech in the many investigations which I have devoted to the subject. Among others I was able to note it in my own children, who learned to speak normally. These little primary awkwardnesses have no special significance; but later, when the child endeavors to express its sensations, opinions, and thoughts, therefore, as soon as the child's vocabulary is sufficiently complete for the expression of its thought, they acquire an immense importance. This expression of thought is the chief characteristic of the *fourth period of speech development*, and if up to this period the power and the cleverness of mimicry have been limited, inhibitions will arise in the form of *mutism* or of *stuttering*.

We are often informed by the parents that at first their child was quick at imitation and could even pronounce a few words, but that after a definite period the words were no longer used and the child has since been almost speechless (deafmutism).

It might be supposed that this condition has originated from futile attempts of the child to imitate the speech of those about it; that in contrasting its speech production with that of others it realized its shortcomings without understanding them, and finally relinquished its efforts in disgust. This explanation is not altogether theoretic, but may frequently be confirmed by an investigation into the origin of these conditions. A *voluntary deafness* of this kind may arise even later, but there will then be more complicated phenomena than in the earlier period of speech development. External factors such as adenoid vegetations, malformations of the palate, etc., may facilitate the development of this condition; in some cases I have found cleft palate to be the cause. Naturally these expositions apply only to normal, intelligent children. If we are dealing with mild or marked idiocy we must expect to find loss of speech and disturbances of articulation. In slight grades of idiocy the general awkwardness is very characteristic, and in the extreme types there will be no speech, for the simple reason, so aptly expressed by Griesinger, that the child "has nothing to say."

The innate instinctive movement of the child may be defective without any special demonstrable mental disturbance. In some cases we may even observe a congenital sluggishness; the child does not learn to walk until late, not because of rickets or other disease, but because it does not desire to walk; there is no great wish to move about and therefore no attempt is made to stand and to walk, as is the case of a normal child. It may not be without signifi-

cance to call attention to a parallelism in children learning to walk and those who are wilfully mute. Children frequently learn to walk early, perhaps at the age of 11 months, but after they have fallen a few times and been hurt they do not make a further attempt until a considerable interval has elapsed—I myself have seen intermissions of three months and longer. If they are left to stand alone they will not try to walk but either sit down at once or begin to cry. It would therefore seem here also that a knowledge of failure of their efforts inhibits the desire for movement.

The second and far more frequent evil which arises from interrupted development and which must be referred particularly to the great disproportion between the motor and sensory speech centers, is *stuttering*. These slight repetitions and numerous trials of speech which with a greatly developed sensory speech center become constantly quicker and more active, may readily produce a spastic articulation, and we observe a characteristic stuttering, sometimes after an evolution of several years, but often in a very brief time.

Finally, the development of certain speech sounds may be more or less retarded so that they appear late or perhaps can never be uttered. To this category belong especially the sounds of the third domain of articulation, *k* and *g*, the fricative sounds *s*, *sch*, and others. These slight errors in pronunciation, which have been included under the collective term *stammering*, are in themselves of no special importance. Naturally, they denote retardation of the motor center in comparison to the sensory. They are only of influence upon the development of the child when they persist for a long time, for example, until the school years.

Children who stutter and stammer are but little conscious of their speech defect; they are almost unaware of their shortcoming which is so apparent to their playfellows. If, however, this consciousness is awakened by accident, or by a change in the training of the child, for example, entrance at school, it has an extraordinarily depressing effect and as a consequence of this depression complete mutism, *aphasia voluntaria*, has not infrequently been noted. Those who even superficially study the development of psychical depression in speech disturbances will generally acknowledge that simple stuttering may lead to mental dejection. That, however, an incorrect pronunciation also produces the same result may often enough be demonstrated in practice. I shall never forget a case which I saw with the late S. Guttman: A boy, aged 7 years, the child of wealthy parents, had been the pet of the family and had received instruction at home. At a children's party which he attended at the house of some friends several boys noted his faulty pronunciation of the *s*-sounds, and with the lack of delicacy and consideration for others which is so common in children, they began to guy him. The lad returned home crying and would not reply to questions as to the cause of his sorrow, but sat quietly in a corner and refused to eat and speak. After a few exercises the little fault was corrected—it was a case of *sigmatismus nasalis*—and the child was as happy as ever. With the removal of the cause the depression disappeared. Every error, particularly of speech, which becomes perceptible to the child



and makes him conscious of his inferiority to his playmates may lead to *symptoms of depression*.

It is quite self-evident that the aforementioned defects cannot be attributed to physiologic phenomena *alone*. Apparently there must be some additional cause which facilitates the development of the evil. For if the physiologic phenomena *alone* were at fault almost all children would acquire defective speech or at least pass through a period of actual speech disturbance before they could speak correctly. Although this conception is not generally recognized, and although it is proper to refer to *physiologic stuttering* and especially to *physiologic stammering* in infantile speech development, nevertheless, for the generation of the pathologic condition some special disturbance is necessary, the nature of which is not always obvious.

There can be no doubt that by far the greater number of children, and especially those who stutter, are of a *hereditary neuropathic predisposition*, and Kussmaul is quite right when he speaks of a *congenital irritable weakness of the apparatus of articulation*—a weakness which betrays itself by speech spasms. Accordingly, those children who acquire stuttering or who have stuttered from the beginning of speech development are, in by far the majority of cases, nervous or neurasthenic. These two conditions may quite properly be differentiated, in that the nervous child reacts more rapidly and intensely to irritation than the normal child; its wave of stimulation lies deeper. The neurasthenic child shows, in addition, an extraordinarily rapid fatigue of all nerve functions, an abnormal, ready exhaustibility of the functionally stimulated nerve regions, whereby the nervous apparatus, even after a conspicuously slight use, reaches a state of irritable exhaustion (see Krafft-Ebing and Erb).

A careful investigation of the *etiology of stuttering* brings us to the conclusion, as Mygind has shown, that the neuropathic predisposition of children is an extraordinarily important causal factor in stuttering. The irritation itself to which such patients react more rapidly than the normal child may be quite obscure to the eye of the investigator. It is true that in children an *endogenous irritation from the intestine* is capable of producing the most astounding nervous phenomena, and the older clinicians well recognized that stutterers frequently suffer from constipation. The irritation of constipation may have a more or less decided influence upon speech provided we are dealing with a nervous individual, and for this reason the old pediatricists advised the administration of purgatives in the treatment of speech disturbances. A comparison herewith of a remark of Romberg in his *Text-Book of Nervous Diseases of Man* (1851) is not without interest. In describing the treatment of stuttering he says: "In treatment attention to the causal indication has in recent years been too much supplanted by gymnastics of the tongue, and here also the futility or even the deleterious effect of the remedial dilettantism is evident. According to the requirement general or local venesection is to be employed, or integrating stimulation, applications of cold, sea-baths, etc., and especially the administration of laxative remedies, which have proved of great service even in the most obstinate cases, of which Bostock has reported a very



remarkable example: Every relapse of stuttering was overcome by the employment of purgatives in combination with a strict derivative diet." (History of a Case of Stammering Successfully Treated by the Long-continued Use of Cathartics. Med. Chirurg. Trans., Vol. xvi, p. 72). The early writers were so familiar with this symptom in stuttering children that the ancient therapeutic régime, proposed by Hieronymus Mercurialis, was purely dietetic. This author, however, did not neglect exercises of speech nor general physical exercise, which he introduced as important measures in treatment. He says: *Exercendum est corpus, quantum fieri potest, praesertim vero exercenda est vox, et si quid est, quod possit prodesse balbis et haesitantibus, est continua locutio alta et clara.*

In the cases mentioned the result of irritation from the intestine was the appearance of speech spasms in the form of stuttering. But this intestinal irritation may also give rise to paralysis of the motor speech tracts, or, better, to an excessive stimulation of the inhibitive tracts, so that articulation becomes impossible and the picture of *aphasia* is presented. Thus Henoch has observed *aphasia from overloading of the stomach*. The aphasia disappeared after vomiting and might therefore be called an *aphasia dyspeptica*. Arndt observed *aphasia after accumulation of feces*, Lichtenstein after *nematodes—aphasia helminthica*. I have seen a case of aphasia in a girl, aged 8 years, which was produced by *oxyuris vermicularis*. The aphasia disappeared after the worms were removed. These conditions were also known to ancient writers and in the Nosologia Methodica of Boissier de Sauvages, among other forms of dumbness we find *mutitas verminosa*: "*Puer ex amphimerina verminosa mutus evaserat; sublata febre mutitas persistit; est post aliquod tempus sumptis anthelminticis triginti sex lumbricos intra vaginti dies deiecit, quibus reiectis loquela rediit, ists tamen ut littera B difficiliter pronunciaretur.*"

Therefore, in all cases of speech inhibition or spasm we must remember that both phenomena may be produced by the same irritation and we must judge in the individual case whether such an irritation is to be assumed, as in the examples which I have mentioned.

Among the endogenous stimulations which come under consideration and which have a direct relation to infantile speech disturbances, we must consider *improper feeding*. Spastic speech defects are particularly common in children of the prosperous classes. In Wiesbaden the statistics regarding the percentage of stutterers among the students attending the Gymnasium showed that there were twice as many cases as were found in the public schools. Apart from the neuropathic predisposition, which we may assume to be in about the same proportion, an improper nutrition is frequently the cause of spastic speech phenomena. I have almost invariably encountered *over-nutrition with meat in nervous* stutterers of the well-to-do. That nutrition of this sort cannot be counteracted by exercises of speech because it maintains the fundamental irritation for speech spasms probably requires no comment. If the nourishment is changed and the child is given a mixed diet, with limitation of meat as much as possible to one meal, the child not only improves in appearance

in a relatively short time, but increases in weight and, what is most important, is more composed. Very little speech exercise is then required to overcome the stuttering. This disappears, we might say of itself, with the elimination of meat, and according to the conception of stimulation and reflex here given, the explanation is simple: The irritation of faulty nutrition is diminished and the reaction of defective speech disappears. A classical example of such cases will be detailed briefly:

A boy, aged 8 years—sent to me by Professor Erb—whose mother was exceedingly neurasthenic, was accustomed to but little variation in his diet. The amount of meat which he consumed in the first three days in my hospital was almost phenomenal; it was sufficient to satisfy a full-grown man. He rejected vegetable food as his mother had taught him that this was difficult to digest. The boy suffered from constipation, was pale, of small stature, and weighed only fifty-two pounds. He stuttered over nearly every word; sometimes he could scarcely speak and had to pause in the midst of a sentence. He was readily annoyed by external impressions. Sounds, noise, the boisterousness of children, were unbearable to him. He was not accustomed to romp and play with other children; his sleep was restless and almost every night interrupted by loud outcries succeeded by weeping.

Before instituting any treatment in the way of exercise I directed my attention to the regulation of his diet. The boy was given food consisting principally of vegetables; the amount of meat was reduced to a moderate quantity at mid-day; vegetables of all kinds were given to him, which up to that time he had not eaten at all or in only small amounts, and he accustomed himself, although at first with some repugnance, to this change in diet. Instead of the glass of sweet wine which he had been allowed at home “for strengthening” (!) and which, naturally, had increased his nervousness, he was given milk. Morning and evening he was bathed in water of the temperature of the room and at half-past seven he was compelled to go to bed. As a result of this treatment the boy gained in weight, soon had rosy cheeks, showed little or no reaction to external irritation, joined in the play of other children, and—*he no longer stuttered*.

When, after a few months, the mother took her child home and asked my advice as to the remedies to be used to prevent a repetition of the stuttering, she was greatly astounded when I handed her the diet list, which consisted of the food already prescribed.

Therefore, dietetic treatment also plays a rôle in speech disturbances that must be well considered. When the condition is properly understood an actual miracle is sometimes effected. In late years, unfortunately, the *clinical observation of children with speech disturbances* has been almost entirely abandoned, so that these conditions are no longer borne in mind by the clinician. This is owing largely to the fact that exercise treatment, which actually plays an important part in speech disturbances of children, and has been considered in another volume of this work,<sup>1</sup> has been forced into prominence and has been

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<sup>1</sup>See volume on “Diseases of the Nervous System,” p. 343.

left to the superintendence of empirics, teachers, quacks, governesses, and other persons who have not been professionally trained. This is a serious error. The slightest defect in articulation which arises during the period of speech development should be investigated by a skilled physician; for only *the reasoning physician can properly investigate all of these individual details, the observation of which is so necessary if the proper results are to be attained.*



# FUNCTIONAL NERVOUS DISEASES OF INFANCY

By H. NEUMANN, BERLIN

In infancy the functional diseases of the nervous system are not merely of great importance on account of their extraordinary frequency; in consequence of the distinctive nature of the infantile nervous system they frequently assume peculiar forms, and their conception in infancy is of importance in their appraisalment to the extent that they usually reach their fullest development only in adults.

Functional nervous disturbances cannot always be precisely defined from organic diseases, nor can they always be accurately systematized. From pathologic states, apparently of a homogeneous nature, different groups of disease often develop, and in the same group the clinical picture may reveal itself in different forms. If we decide to consider certain signs of seemingly increased nerve activity only as the expression of an irritable weakness, we may make a schematic division by regarding some diseases as phases of a functional debility, others as an expression of an abnormal course. To go further, we may make a distinction between affections of the nervous system and mental diseases. If we leave the latter out of consideration, we must contrast neurasthenia with hysteria among the functional nervous affections. But in hysteria, particularly when its starting-point was in infancy, we often meet with conditions which cannot as yet be differentiated from those of neurasthenia.

In some respects Graves' disease, epilepsy, and chorea may be regarded as transitions to the organic nervous affections.

A numerical report of the occurrence of functional nervous diseases in infancy is difficult to make; the clinical material does not present itself alike to every observer, and many disturbances, if they are mild, are unquestionably overlooked. As these objections are comparatively less obvious in my dispensary practice, the following statistics are given: in the years from 1894 to 1902 there came to my Polyclinic<sup>1</sup> (Dr. S. Kalischer), besides 397 organic diseases, 741 functional neuroses and psychoses. Notwithstanding the fact that nervous diseases were also seen at other times than in the nervous clinic, there must be added to the former number over 1,000 cases of spasm of the glottis, tetany and convulsions (essentially on the basis of the latter). Therefore the overwhelming majority of functional neuroses in infancy belongs to the realm

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<sup>1</sup> See Fritz Schiffer, *Jahrb. f. Kinderhk.*, 1904. The cases referred to later are not limited to these alone, but are also taken from private practice.

of tetanoid hyperirritability of the nervous system, that is, of simple convulsions. Next in number are conditions of mental weakness (in our statistics the minimum was 100), and of irritable weakness (minimum 94). The importance of the latter—neurasthenia or nervousness—becomes all the more apparent if we include the following groups: Headache and migraine (97 cases), *maladie des tics* (23 cases). In contrast, the following diseases are far behind in frequency: epilepsy (118 cases), hysteria (71 cases), spasmodic nutans (25 cases). Finally, chorea must be mentioned, with 122 cases, and Graves' disease, with 32 cases. The number of cases of perverse psychic development cannot be reckoned exactly; psychoses—more frequent after the period of infancy—came under observation only 16 times.

### CONDITIONS OF IRRITABLE WEAKNESS

**Convulsions.**—First, on account of their obvious importance and exceedingly common occurrence, we shall consider the *conditions of irritable weakness* of early childhood. The best-defined and most serious manifestation of this weakness of cerebral function is *convulsions*. It is true, they are no longer classified as a substantive affection (eclampsia), but merely a symptom of disease. In another part of this work an explicit description will be given of these conditions. Here but a few remarks are necessary, as the convulsions which originate from disease of the central organs and their membranes will not be considered. The convulsions which occur in the course of severe affections, for example, cholera infantum, and which are to be explained in a manner similar to uremic convulsions—as symptoms of intoxication—will also be omitted. I shall only refer to the tendency to convulsions which are most pronounced in the newborn in conformity with the undeveloped nervous system, and which become rare with advancing age. These are observed particularly in dyspeptic disturbances—from the transitory contractions due to flatulence, to the convulsive seizures which last for days with intervals of but a few minutes and which may occur even in breast-fed infants. The tendency to convulsions, incidentally with sudden high fever, lasts a comparatively long time in the infant and in the adult these are supplanted by chills. I have observed these febrile convulsions most frequently at the onset of croupous pneumonia,<sup>1</sup> rarely in influenza, diphtheria, parotitis or other diseases; they have not occurred in my practice later than the fifth year, but Henoch reports the condition in children of much older age. In this connection attention must be called to a special form of convulsions in which there is *arrest of breathing*. This apnea or “stoppage”—which is not to be confounded with spasm of the glottis—is most common in the second year, infrequent in the third and fourth years, and is probably somewhat more general in boys than in girls (8 to 5). Following a blow or a fall, punishment, anger, or a paroxysm of rage, there is

<sup>1</sup> Many of them are not the results of high temperatures, but of pneumococcus affection of the brain. The germ is mostly eliminated through the kidneys.—EDITOR.

an arrest of respiration, the child becomes blue and rigid, and is attacked by clonic convulsions; in a few moments it breathes again, either voluntarily or after shaking, slapping, or sprinkling with water.

The functional nature of this entire group of convulsions is evinced by the absence of successive disturbances of the nervous system. As a rule they arise the more readily the younger the child and the greater the irritation. When both of these factors are considerable, possibly the neuropathic tendency, as a predisposing agent, may be of less importance. In many cases, for example, of apnea and also of febrile convulsions, it is exceedingly easy to prove not only the predisposition but also the presence or later development of neurasthenia and even of typical hysteria, and for this reason the susceptibility to convulsions is mentioned at this point.

I shall be even more brief in regard to the group of convulsions which occur in tetanoid hyperirritability of the nervous system and which have recently been much studied. Although they have a familial tendency, like the group of convulsions just described, the neuropathic heredity is not so distinct. They appear under the influence of the same deleterious factors of respiration and nutrition which act upon the development or exacerbation of rickets. Correspondingly they preponderate in the first half of every calendar year.

It is immaterial whether we are dealing with an apparently substantive eclampsia or with spasm of the glottis, perhaps succeeded by general convulsions, or whether we are dealing with the typical convulsions of tetany, the fundamental structure is always an increased irritability of the nervous system which may readily be demonstrated by the motor and electrical tests of the peripheral nerves; this condition may also remain latent—without convulsions. It is so extraordinarily common in infancy that, more than any other form of convulsions, it impresses upon the delicate age of childhood the stamp of spasmophilia.

**Nodding Spasm.**—A much rarer form must be mentioned, i. e., *nodding spasm* (*spasmus nutans* or *nictitans*). The principal symptoms are tremor of the muscles of the neck and peculiar movements of the ocular muscles; nystagmus in one or both eyes (lateral, vertical, or rotatory), with an occasional decided lateral fixation. Without entering upon a minute description of this remarkable affection I may state that it has no connection with tetanoid hyperirritability of the nervous system. Nevertheless, its periodic appearance follows laws similar to the other forms of spasm. Thus, among 35 cases, I observed 31 between December and April and only one in May, June, October and November respectively. Complication with rachitis is not typical, therefore we may assume that the same deleterious effect of season, as in rickets, also favors the appearance of *spasmus nutans* as well as tetany. Nodding spasm may occur until the end of the third year. It appears most frequently between the sixth and eighteenth months (first six months 7 cases, second six months 12, eighteen months 14, two years 5, two and a half years 4, three years 1). The preponderance of females is conspicuous (28 girls, 15 boys).

If we disregard spasm as a definite manifestation of irritable weakness and



attempt to describe the gross appearance of pathologic pictures, we meet with certain systematic complexities. Hereditary degeneration may become distinct in a neuropathic as well as a psychopathic sense. A congenital neuropathic constitution produces phenomena that may coincide largely with those of neurasthenia due to various causes, as onanism (even in childhood); the most varied phenomena of irritable weakness may be present in either case. In any event some of the symptoms which occasionally occur with a neuropathic degeneration are usually lacking in acquired neurasthenia. Further, we are concerned with typical neurasthenia which not only takes the form of nervous exhaustion, but simultaneously produces a peculiar mental condition whereby the neurasthenic subject "is distinguished by a tendency to a constant subtle analysis of his condition, by permanent sensations of fear, and by lack of will"<sup>1</sup>; in this sense neurasthenia of well-characterized type is comparatively rare in children. A differentiation of these various forms of neurasthenia has been found to be practically impossible. We shall therefore treat of **nervousness** or **neurasthenia** (used synonymously) as a great and essentially uniform pathologic group.

This, however, does not dispose of all of the difficulties. On the contrary, before entering upon our theme an attempt must be made to ascertain the relation of neurasthenia to **hysteria**. These conditions are very frequently combined; as to their development, the symptoms of hysteria usually appear progressively at a time of nervous degenerative weakness—seldom during the first years. In the literature we find two descriptions: Some authors set forth neurasthenic symptoms, for example, nocturnal incontinence, night terrors, as the tokens of neurasthenia, and infer that the hysteria appears in addition. Others regard these conditions as the first concealed expression of hysteria ("*hystérie naissante*") of St. Philippe<sup>2</sup> or as a distinct expression of hysteria of early childhood which has not yet been correctly appreciated (Bézy-Bibent,<sup>3</sup> Thiemich.<sup>4</sup>).

It might be supposed that with a clear definition of hysteria a conclusion could be reached in every case, but this is extremely difficult, especially in childhood. The earlier explanation of Möbius, that all of those morbid changes of the body which are limited to the imagination are hysterical, is inadequate even for the hysteria of adults, as is also the later amplification, that some of the hysterical symptoms are not altogether suggested but are a pathologic reaction to emotional agitation. Neurasthenia may also be the result of a pathologic reaction. We must agree with Jolly<sup>5</sup> that "in addition to the alteration of emotional and reflex excitability the psychic symptom of an increased imagina-

<sup>1</sup>Theodor Dunin, "Grundsätze der Behandlung der Neurasthenie und Hysterie." Berlin, 1902.

<sup>2</sup>Saint-Philippe, Art. "Hystérie; Traité des Maladies de l'Enfance," Tome IV, Paris, 1898.

<sup>3</sup>"Die Hysterie im kindlichen und jugendlichen Alter." Translation by Brodtmann, Berlin, 1902.

<sup>4</sup>Jahrb. f. Kinderheilkunde, N. F. VIII.

<sup>5</sup>Berliner klin. Wochenschr., 1892, Nr. 34.

tive faculty" is introduced; the "actual hysterical phenomena, a true hysterical symptom-complex, and the entire clinical picture of hysteria, appear only when this effect of the imagination is combined with the symptoms arising from the corporeal (nervous) condition." If we agree with this view and accordingly decide the neurasthenic or hysterical nature of a disturbance, we must still determine whether the diagnosis of true hysteria—we often speak of hysteroid symptoms—may be made at once from the action of every abnormal imagination incidental to neurasthenic conditions; for the imagination is sometimes so greatly developed in children that there may be a transitory slight aberration.

In connection with this question is a further one: Whether the success of suggestive therapy is such as to verify the diagnosis of hysteria in every case. If the part played by the imagination is so great in infancy as is generally recognized, if, further, the unconscious as well as the conscious psychic life can be influenced with comparative ease, there will be, under some circumstances, as the innervation of an organ, a successful regulation by suggestion, which, however, does not prove that the condition is hysterical. Artificial emphasis of a conception need not be such, even when it leads to the repair of a pathologically changed function, as to force a pathologic conception aside; whether the conception acts consciously or unconsciously need not be taken into consideration. Here is an example! The voluntary discharge of urine is inculcated in the child by certain external circumstances: restraint is brought about by education, which eventually results in an automatic reflex process. If the inhibitive apparatus is not sufficiently powerful, or if the stimulation in the urinary organs and the corresponding spinal reflex are too active, a discharge of urine occurs involuntarily. Under some conditions a normal condition will be brought about by stimulation of the will or incitement of the inhibitive apparatus to greater function through a mediate influence; this stimulation may be psychic, as in treatment of hysteria—nevertheless, we are not dealing with hysteria. We would have to do with the latter affection if the evacuation were the result of a hysterical conception; for example, if it were thought impossible to control micturition under certain external conditions, recovery might be brought about by the removal of this idea. Therefore, the same psychic influence might be active under various psychologic causes—by indirect strengthening of the will and by the removal of the pathologic impression. The minute analysis of the method of cure will frequently decide whether we are dealing with neurasthenia or hysteria. A combination—*neurohysteria*—would be present in our illustration if, as the cause of the conception, there were an actual disproportion between reflex irritability of the bladder and the energy of the inhibitive apparatus, so that, under certain transient and local conditions, micturition could not be controlled.

Therefore, we shall differentiate between pure neurasthenia, pure hysteria, and *neurohysteria*.

The portrayal of the chief characteristics of the nervous child must necessarily be crude, partly because personal experience is naturally limited and the

space is insufficient for an exhaustive exposition, and partly for the reason that neurasthenia would *not* enter into consideration if the nervous disturbance were a part phenomenon of some other definite disease, for example, tetany, chorea, or Graves' disease.

**Neurasthenia.**—In infantile neurasthenia the early symptoms are those of an abnormally mild onset and an abnormally marked efflux of the nervous reaction which are covered briefly by the term "nervousness." As a rule we have to do with a congenital irritable weakness, therefore a careful observation will often show very early signs of the affection. The first symptom is an abnormal reaction to impressions which are transmitted by the organs of special sense and the sensory nerves of the skin. Furthermore, in children beyond the age of infancy the organs of special sense (for example, sense of smell) often show an excessive sensitiveness to impressions sometimes below the wave of normal. Even in earliest infancy sudden noises, perhaps when moderately loud, will produce fright and trembling, although the latter symptom is less common.

With progressive inclusion of the central organ in the reflex tract the sensory stimulations and those of the special senses produce not only abnormally powerful reflexes but an over-active and at the same time strongly fluctuating, psychic irritation. Children may show a nervous predisposition by their great activity at play and by the conspicuous involvement of their imagination; pleasant or unwelcome slight incidents elicit exaggerated expressions of pleasure or of disgust. Often there is great capriciousness, with a lack of discipline this may develop into obstinacy, and a conscientious and earnest attempt to combat this condition gives rise to greater excitement; the above-mentioned apneic attacks appear in the first years of play and are partly the result of anger or rage.

The nervous child is not only active but is readily excitable, irritable, and sensitive, and thus a painful disturbance of the emotional equilibrium may be brought about by some slight external cause. The incapability of normal response to the influences of the outer world and of their orderly and quiet utilization often causes anxiety of mind, so that the child is shy and cries upon the slightest provocation. Even older children who have been deprived of their mental equilibrium by fright—often by some mummery or stupid joke—may continue in a prolonged state of terror, so that they cannot sleep in a closed or dark room, or without the mother's presence. More individual, constrained illusions of fright, partially hereditary, are also noted, as a fear of blood, of dogs, of worms, and the like. Sometimes a child will not stay alone in a room nor walk alone upon the street, will not ascend steps nor go into the cellar. As an example of complicated *phobias* I must mention the case of a boy, aged four years, who, frightened generally at everything new or sudden, out of doors was afraid of the wind, later of a storm, and would cry "Don't hurt me, wind, don't hurt me!"

The periodic relaxation of the nervous system, so necessary for normal development, in the nervous child is often insufficient in duration and intensity.



Next to abnormal emotional excitability disturbances of *sleep* belong to the most common symptoms. *Insufficient sleep* must first be mentioned: The child falls asleep late, less commonly it awakens too early or will lie awake for hours at night; it either rests quietly with open eyes, plays, "romps," or talks to itself or to those in the room. Young children sometimes sleep only when sung to, or when they are well covered and thus restricted in their movements. While the nervous infant is wakeful, the sleep of older children is usually disturbed, and the increasing number of illusions which are associated with more or less decided sensory impressions are retained even in slumber. The child tosses about and talks of things connected with the events of the day; occasional cries, screams, or moans betray the action of unpleasant and annoying psychic processes.

**Night Terrors.**—A few words must be devoted to *night terrors* (*pavor nocturnus*) although some authorities do not include this disturbance among the symptoms of neurasthenia. Under some circumstances there may be confusion with an epileptic equivalent, while the origin of night terrors from hindrance of nasal respiration, from worms, or from debilitating influences by no means precludes a neurasthenic affection; a minute examination will always reveal other signs of nervousness. Night terrors are to be looked upon as a profound nervous disturbance of sleep. They may continue for a protracted period, or periodically or only occasionally. A short time, perhaps an hour, after the child falls asleep, it sits up suddenly, or jumps out of bed, glances around fearfully, cries and screams at terrifying hallucinations, and is only gradually quieted, perhaps after a quarter or half an hour, by the turning on of the light, by talking, or by being taken into the mother's bed. A second attack may occur in the same night, or the sleep will be restless. Forgetfulness (*amnesia*) closes the door on these nightly occurrences.

In my experience night terrors appear earliest in the third year of life, are of about equal frequency up to the tenth year, and from that time they become rare (from the third to the fifth year 9 times, sixth to tenth year 14 times, eleventh to fourteenth year 6 times; 17 boys, 12 girls).

We now come to the mobility of the nervous temperament. Not rarely it is revealed very unfavorably by a tendency to jump, flightiness, inattention, and may find its most conspicuous aspect in *great physical unrest*. The child is only transitorily quiet; to use a common expression it is fidgety, "like quicksilver." Even when the child tries to be still, or when its attention is arrested, the respite is only for a few moments. This abnormality may disappear in time, while its increase necessitates an unfavorable prognosis as to cure, since alternate improvement and aggravation of the condition may persist indefinitely. In the latter case we speak of *maladie des tics convulsifs*, a combination, as it were, of numerous varieties of tic, i. e., apparently voluntary, but actually constrained and oft-repeated movements.

**The Tics.**—Often there are movements of the legs and of the lower trunk; but the hands are seldom quiet, they are passed over the face, the back is rubbed, the nose pulled, the sexual organs handled. Of greatest frequency are

movements in the musculature of the shoulder-girdle, of the neck, and of the face. The shoulders jerk, the head is shaken, turned or otherwise moved, the angle of the mouth contracts, the lips twitch, there is blinking, winking, the eyebrows are raised and lowered, the forehead is wrinkled. There is snuffling, hawking, and in rare cases there are peculiar exultant or inarticulate sounds, but I have never heard the forcible utterance of obscene words such as are forthcoming from adults. Every patient has his individual combination of movements which recur for some time. These enforced movements appear rarely before the fifth and most frequently after the eighth year of life; the sexes are affected equally (in the fifth year one case, from the sixth to the tenth year 19 cases, from the eleventh to the fourteenth year 13 cases; 17 males, 16 females).

The transition to *simple tic*, which will not be described here in detail, is brought about by constrained movements, still in part physiologic, which are merely designated as *bad habits*. *Sucking of the finger* belongs to this category; in small children the skin and the nail of the finger become macerated and in later years the finger-tip may be calloused, the phalanx flattened, the nail broad and somewhat concave. While sucking of the finger is probably first brought about in the new-born by the accidental introduction of the finger into the mouth or by hunger, it finally becomes a habit, which is particularly indulged in just before sleep, but is practised also in sleep and at other times. As a rule the habit is abandoned at the school age. *Biting of the nails*, together with degenerative phenomena, is much more common.

The tendency of young children to put *foreign bodies* not only into the mouth, but occasionally also into the other natural orifices of the body, may develop actual disease in nervous individuals. Thus, a girl aged thirteen months, who presented some nervous symptoms but who was well-developed mentally, placed wood, pieces of candle, and the like, in the nostrils and mouth, ate wool and paper, and even swallowed her feces—an exceedingly rare occurrence in sane children. (A proper education finally conquered the habit.) Another infant, after learning to creep, ate the plaster from the wall and swallowed many foreign bodies, and when two years old the mother pulled a piece of twine out of the anus. Later this child picked off and chewed wool from his clothes, the bed-covers and portieres; at the age of nine years the boy was extremely nervous and only of average mentality.

Certain *constrained processes* must be included here which commonly occur only in *sleep*, although under certain circumstances, like some involuntary movements of hysteria, they are observed during complete consciousness. Transition to these also may occur from movements yet within the scope of the norm. Many children will fall asleep only when their finger or a portion of the bedding is in their mouth, when the bed-clothes are drawn over the face, when they are uncovered, or when they are sitting, or lying upon the belly. Close to the norm also is the habit of some children of *gritting the teeth*; its sudden appearance, however, arouses a fear of disease of the brain. *Cough* during sleep every night at a certain time is not uncommon and is sometimes



associated with retching and vomiting which naturally awakens the child. The fact that cough is absent while the child is awake and can be overcome by suggestion indicates its nervous nature.<sup>1</sup> *Automatic movements* in sleep are various; they are usually noted in children with grave hereditary degeneration. I saw a girl aged nineteen months who almost every night, before midnight, began to kick and scream. Later the child suffered from pseudo-croup, her sister from hystero-epilepsy; subsequent to my visit the condition disappeared suddenly, after a continuation of nearly six months. A girl somewhat older chewed and swallowed in her sleep and executed rhythmical movements with her elbows. Other children sleep for hours upon their belly, with the head pressed against the pillow, and perform rocking movements with the trunk which are at times so severe as to shake the bed.

The classification into a particular system of disease of the *dream actions* which we observe in children may sometimes be exceedingly difficult. Children with vivid imagination have hallucinations in sleep in which they continue their play. Other children speak in their dreams of school or of the occurrences of the day. They may even rise and perform complicated movements, such as packing their school-books. Complicated somnambulistic processes lead from the realm of neurasthenia to hysteria.

**Onanism.**—In conclusion, *onanism* must be described; this is legitimate in so far as it is an impulsive action of nervous children. Onanism in older, normal children, which is the result of the example or was taught by servants, rarely comes to the notice of the physician. In the worst cases, as already mentioned, it may lead to severe neurasthenic exhaustion.

*Onanism* is practised equally by both sexes. My statistics show, during the first five years, 6 boys and 9 girls; from the sixth to the tenth year, 10 boys and 11 girls; from the eleventh to the thirteenth year, 4 boys and 2 girls; a total of 20 boys and 22 girls. The beginning of the vice may often be traced to early childhood (in 42 of my cases the habit began in at least 18 instances in the first five years). In the first two years onanism appears to be more common in girls (among 9 children of this age 6 were females)—a fact already mentioned by Hirschsprung.<sup>2</sup> In little girls onanism is often overlooked: the child, in my experience, at the earliest six months old, suffers from peculiar attacks in which the legs are pressed tightly together, or more rarely are crossed, and the hands are sometimes folded: there is pressing, squeezing, and groaning, the face is flushed, and there is an outbreak of perspiration. The act of masturbation cannot always be interrupted even by raising the child from the recumbent posture. One of my cases, a girl aged 9 months, drew herself toward the bed-post and, with pressure and groans, stamped with her legs. Older children perform swaying movements while seated, sometimes accompanied by singing. Occasionally they slide along the floor, or lie upon the abdomen and perform rubbing motions. In children of still greater age

<sup>1</sup> A cough appearing after sleeping demands the examination of the throat (chronic catarrh, large tonsils, long uvula).—EDITOR.

<sup>2</sup> *Berliner klin. Wochenschr.* 1886, Nr. 38.



the movements are definite, especially friction upon the genital organs with the hands.

I need not dwell upon the persistence of onanism at every period of the day. Masturbation is practised at night in half sleep as well as while awake. There are sexual equivalents for onanism in children of one to two years who, after weaning, suck their fingers, pieces of linen or the bed-clothes, not infrequently, as Steine<sup>1</sup> reports, "with simultaneous erection, marked flushing of the face, unusual brightness of the eyes and, finally, with an outbreak of perspiration." This most commonly occurs while falling asleep or shortly before awakening. The same symptoms are noted in nurslings. I learned that for three years a patient of mine, a boy aged 10 years, had sensations of lust when he watched flowing water or snakes in motion, and that while dreaming of similar things in his sleep he would lie upon his abdomen and involuntarily produce friction. Such indefinite sexual irritations—especially in sleep—are so common during pubescence that they can then scarcely be regarded as pathologic.

The consequences of persistent onanism in young children are comparatively insignificant, and consist mainly of exaggerated irritability which may result in attacks of mania. Older children who are addicted to this vice are shy, disagreeable, sullen, downhearted and stubborn; they are slow to learn, are languid, have dark circles around the eyes, and complain of palpitation. The patellar reflex is active, the penis is readily erected; various nervous disturbances, such as the *tics* may also be present. Irritation in the vicinity of the sexual organs may be the cause of masturbation, such as oxyuris, or chronic eczema of the genitalia and of the thighs, which produce pruritis. Accidental pressure during the discharge of urine and feces may be a first cause and perhaps explain the more frequent occurrence of masturbation in little girls. The prognosis is least favorable when onanism is practised by small children in the absence of a recognizable cause.

The foregoing deals in the main with the abnormal development of the psychic life of the nervous child under the influence of the external world; more difficult is the description of the uncommonly diverse influences exercised upon the nervous system by the sensations which originate from the different organs of the body. Briefly stated, the sensations arising in various directions from a pathologic condition may produce abnormal impressions upon the sensory sphere, and, under certain circumstances, influence the will and conception unconsciously, so that transitions to hysteria arise; furthermore, the reflex processes of muscular and vasomotor nature are variously influenced abnormally. The appreciation of these conditions is rendered difficult because we simultaneously have to deal with disease of a peripheral organ (under some circumstances of the brain itself), and a central nerve which reacts pathologically, and a correct estimate of the part played by each component cannot always be made at once.

The nervous child may have, from insignificant causes, for example, dis-

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<sup>1</sup> "Compend. der Kinderkrankheiten," 3 Aufl., Leipsic, 1878, p. 288.

turbances in the digestive tract—attacks of screaming which are unusual in their severity and duration and make those about him almost frantic; under some circumstances they may lead to reflex convulsions. On the other hand, severe debilitant conditions may occur without serious organic disease. I remember the case of a child, aged five months, who had periodic attacks of “insane” screaming, particularly at night, and, as I convinced myself, could scarcely be appeased after some trifling annoyance, but when nursed it succumbed “with lusterless eyes.” The feeling of weakness, of lassitude, occasionally associated with somnolence and early fatigue when at play, and especially during study, is particularly common at the school age, and is then maintained by the various causes of neurasthenia (school, disease) which will be considered in detail later. This may be associated with a melancholy or peevish disposition, perhaps characterized by frequent sighs or thoughts of death; sensations of syncope are unusual but vertigo is common.

**Vertigo.**—In many children the sensation of vertigo appears after great activity of movement, therefore, they cannot endure to ride in a carriage or upon electric or steam railroads. This symptom is often accompanied by vomiting. I do not know whether such children are especially subject to vertigo when looking from a height, or to seasickness.

**Headache and Migraine.**—Headache is extraordinarily common in the nervous individual and must be discussed somewhat more in detail. Although it is probably due most often to vasomotor disturbances, its nature is by no means clear. *Migraine*—that form of headache which appears periodically—is not rare in infancy, although customarily it appears much later. I do not altogether concur in Oppenheim’s statement<sup>1</sup> that the usual development of the affection is at puberty. In 9 of my 43 cases it began between the second and fifth years (once in the second and once in the third year), in 21 cases between the sixth and tenth years (10 times after entrance at school), and in 13 cases between the eleventh and fifteenth years. Neither do my figures correspond with Oppenheim’s report as to sex, namely, that females are more frequently affected; possibly after childhood (with which only we are concerned) the sexual life of woman is more productive of migraine. Among my 43 cases 27 occurred in males and only 16 in females. In the first five years of life both sexes are equally affected (5 males, 4 females). From the sixth to the tenth year the male sex predominates (14 males, 7 females). From the eleventh to the fifteenth year the proportion was 8 boys to 5 girls; therefore, notwithstanding their dawning maturity, females are not in the majority even at this age.

Migraine is a typical example of homologous heredity, scarcely equalled by any other nervous disease. Only 5 of my 43 cases were lacking in this respect. In 26 cases (therefore 60 per cent.) the mother suffered from migraine, in 16 instances the father, both parents in 3 cases, and 3 times other ancestors. In some instances brothers and sisters or several members of the

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<sup>1</sup>“Lehrbuch der Nervenkrankheiten,” 1902, 3 Aufl., p. 1010

family suffered from the malady. The well-known typical symptom-complex is usually present even in childhood. As a rule, the headache is localized in the frontal region; rarely, as I found in coincidence with Henoch, is there a well-developed unilateral affection. Pain is seldom present in the occiput; vertigo, nausea, and vomiting are usual. The attack terminates in sleep, from which the child awakens well. The duration and frequency vary as greatly as in later life. As a rule the cases are of the angiospastic type of migraine, with marked pallor; rarely there is an alternation between pallor and flushing. Sweating is common. The eyes are seldom involved (usually flitting scotoma or loss of vision); a hypersensitiveness to noises is noted. There may also be simultaneous abdominal pains with frequent evacuation of urine and feces. Under rare circumstances a trigeminal neuralgia may replace the headache; the attacks are sometimes incomplete or may pass into a more or less continuous headache. In quite a number of children who suffer from migraine nervous symptoms are demonstrable; while many of them may show fair mental development, others are distraught and of poor mentality. An outbreak of migraine is often brought about by attendance at school (conspicuously often in boys!). Often during the holidays there is a cessation of the attacks.

**Nervous Headache.**—No less important than migraine is simple *nervous headache* (53 cases, as opposed to 43 cases of migraine). It is rarely paroxysmal but continues for hours or days or through the night. Occasionally it is associated with vomiting or vertigo. As a rule it is due not only to mental or emotional exertion but to other causes as well. This form of headache, like typical migraine, frequently begins in early school life, and from that time it increases, but is generally absent when the child does not attend school, as during the holidays. Study, writing, singing and gymnastics are sometimes mentioned as etiologic. The pain is increased by heat, running, and the like. The affection may occasionally influence the power of thought and memory. While the factors just mentioned are capable of producing the affection, other causes are injury to the head, inflammation of the brain, and infectious disease, as scarlet fever. A conspicuous cause is disease of the ear. On the other hand, this form of headache frequently occurs in children with the most varied neurasthenic symptoms; in particular a large number of such children are of feeble mentality and therefore are not equal to the requirements of school, and another very large group are afflicted physically, above all scrofulous.

Sänger<sup>1</sup> calls attention to a form of headache which is usually localized in the frontal region above the eye-brows and is to be regarded as an additional symptom of *nervous asthenopia*. I have little knowledge of it.

The distribution differs as to age and sex from that of migraine; in the first five years<sup>2</sup> 4 children (the youngest two years old, 2 males, 2 females), from the sixth to the tenth year 16 children (7 males, 9 females), from the eleventh to the fifteenth year 32 children (20 girls, 12 boys: explained, perhaps, by pubescence and chlorosis).

<sup>1</sup> *Neurasthenie und Hysterie bei Kindern.* Berlin, 1902.

<sup>2</sup> Time of observation.



**Gastric Colic.**—In children *nervous "gastric colic"* is much less common than "*head colic*." The condition should only be considered as the cause of abdominal pain after careful investigation has excluded all organic affections. As a matter of fact when there is no attack the examination of the abdomen does not elicit pain. Occasionally the intestines may be palpated as narrow, tightly contracted strands and the abdominal aorta is felt to pulsate with unusual force. The attacks are most frequent at night. The severe abdominal pains (usually in the region of the scrobiculus cordis) are associated with nausea and vomiting; the child cries or whimpers, the legs are drawn up, and pressure is made upon the abdomen. Heat and particularly pressure and friction over the belly ameliorate the condition; the ingestion of food results in some cases in a renewed attack. In the intervals between the seizures the appetite does not appear to be disturbed. I have observed these colics mostly between the sixth and tenth year of life and somewhat more frequently in girls. Upon careful inspection other nervous symptoms will be found.

Attacks of head and abdominal colic, indicating an extensive vasomotor disturbance of innervation, are rare. A pale, nervous girl, aged 12, who had a small goiter, suffered for a year from head and abdominal colic, with syncope and chilliness, in which the face was pale, the hands cold, and there was formication; after a duration of about an hour the attacks gradually subsided.

**Local Asphyxia.**—Certain varieties of *local asphyxia* must be mentioned, as well as the secretory disturbances which are especially distinguished by local or general *sweating*. (Other forms of asphyxia, for instance, when they are associated with paroxysmal hemoglobinuria, are to be referred to syphilis.)

**Bronchial Asthma.**—At this point, contrary to general custom, *bronchial asthma* will be considered. It may readily be differentiated from hysterical, cardiac, and uremic asthma. An infectious catarrh of the respiratory passages, whether it be true influenza or some other infection, not only produces a gradual swelling of the mucosa but simultaneously a spasm of the smooth muscles of respiration. The catarrh may have been acquired recently by contagion—as is usually demonstrable—or may be of long standing and flare up when opportunity offers; the recent catarrhs are productive of asthma, especially in the cold seasons of the year. With marked involvement of the larynx the symptoms-complex of *pseudo-croup* is developed, but if the deeper respiratory passages are involved the clinical picture is that of asthma. The latter condition need not be especially described here; nevertheless it should be mentioned that while the adult gasps for breath in the sitting posture during the attack, the little child may lie upon the abdomen or upon the side. Asthma, like pseudo-croup, commonly occurs at night. After the nervous outbreak is past a catarrhal condition of the respiratory passages becomes prominent and runs its course with or without complications. Boys are more often affected than girls (in the proportion of 16 to 11). The initial attack is often in the first years of life. In so far as its onset could be determined, among 28 cases asthma began 17 times in the first five years, and of these, 5 times in the first and 3 times in the second year. Careful investigation will always disclose a

nervous predisposition; occasionally there is a directly homologous heredity. Just as in many families migraine is prevalent, so also is asthma. The children show the symptoms of neurasthenia in the most varied combinations; not infrequently they suffer from urticaria or from chronic eczema.

Whereas asthma may be referred to an abnormal reaction of the smooth musculature, in *nervous vomiting* and *nocturnal enuresis* there is a combined action of the voluntary and involuntary muscles. In contrast to asthma, both of these symptoms belong to the most common manifestations of infantile neurasthenia.

The *ingestion of food* by the normal infant is often frustrated by psychical influences; the child is disturbed in nursing by anything that may divert attention—playthings, noises, strange people. The change from fluid to solid food is very difficult to accomplish. Special preferences in taste are also noticeable; food that is not desired or that is forced upon the child is vomited. In the nervous subject these conditions are often developed to a pathologic degree. In certain cases drinking is hindered by the child's activity; there is great disinclination for solid food, and slowness in chewing and swallowing. A bolus of food may be kept in the pouch of the cheek for several hours. A single meal occupies hours and still is insufficient. Deglutition, provided it occurs at all, is brought about by retching, and is succeeded by vomiting. As every variety and degree of difficult nutrition may be due to nervous influences a judgment of the actual condition is not always easy. In addition is the possibility that the transition from nervous anorexia to a fundamental disturbance may be unnoticed. According to Pawlow's investigations the secretion of gastric juice is governed essentially by psychic influences, by the desire for food. Therefore in physiologic gastric digestion there is a psychic element which may be opposed by a disturbance of the central nervous apparatus as well as by defects in the digestive canal. A participation by both components is probably not rare, although which of them is the first to suffer functionally cannot always be determined.

The repugnance to food and the subsequent vomiting can more readily be judged aright if they have to do only with certain articles of diet, and after they are vomited the child ingests the remainder of the meal without difficulty. Emesis of this sort occurs readily in children; even the sight or smell of something obnoxious will give rise to it.

Every conception which is associated with marked emotion is liable to produce vomiting in a nervous child. It is well known that such children vomit in the morning before going to school, but never on holidays nor during the vacation. When breakfast is eaten without appetite and in insufficient quantity the food which has been forced into the stomach may readily be the original cause of emesis. This occurs after breakfast, usually on the way to school, rarely in the school-room, without any special after-effect. From this condition there are transitional processes to hysteria, but true hysterical disturbances of the digestive canal will be considered later.

**Enuresis.**—The voluntary retention or *evacuation of urine* is a complicated

act of the will; not until toward the end of the first, and often in the course of the second year of life, does the child learn to control this function, and in those whose physical growth has been retarded—to say nothing of the children who have not developed mentally—a longer time may elapse. Just as the static functions which have been acquired *latest* are forgotten after debilitating diseases may the control of micturition in early infancy be again lost through an enervation of the nervous system—therefore, after any somatic affection or from excessive psychic irritation, for example, a residence at the seaside. In nervous children also the simple diversion of play, any excitement, fear or sudden fright, may result in an involuntary discharge of urine, and, exceptionally, also of the feces. Even slight distension of the bladder will incite an involuntary discharge, either through an insufficient innervation of the sphincter vesicae or an abnormal reflex stimulation of the detrusor muscle; the latter cause is indicated by the spastic condition (with increased patellar reflexes) which is often demonstrable in the lower extremities. Furthermore, the irritable bladder causes frequent voluntary evacuation, sometimes every few minutes (*pollakiuria*); if this act is not performed at once the urine is ejected voluntarily. Very often this accident occurs without any attempt to control. In rare instances the involuntary expulsion is not periodic but occurs as a constant dribbling. In the majority of cases, in addition to *enuresis*, *diurna*, or *pollakiuria*, there is nocturnal evacuation (*enuresis nocturna*); less often the latter condition alone is present. In favorable cases *enuresis nocturna* may be prevented by awakening the child for the voidance of urine, particularly when the expulsion can be expected at a definite time after the child falls asleep.

From this explanation it is evident that involuntary micturition occurs most commonly after the second year, i. e., between the third and fifth years, and after that period is gradually overcome. Males are more often affected, at least in infancy. I have observed 25 cases of *enuresis* between the second and fifth years (14 males, 11 females), 19 cases between the sixth and tenth years (15 males, 4 females), 5 cases between the eleventh and fourteenth years (2 males, 3 females)—a total of 31 boys and 18 girls.

Careful investigation, at least in older children, may disclose other neurasthenic phenomena of varying degrees. If this evidence cannot be obtained we must determine whether the *enuresis* is due to other nervous affections (idiocy, paralysis) or to local causes (catarrh of the bladder, calculus, etc.).

Various localized neuroses might be included here, such as writer's cramp. *Stuttering* is of special importance, but a comprehensive description of the condition may be omitted.

Having described the individual symptoms of pure infantile neurasthenia, an attempt must be made to classify them according to the age at which they appear. In the first, but more frequently in the second, year great nervous irritability becomes conspicuous (abnormally active reflexes from sensory impressions); the onset is gradual even in the emotional sphere. Active bodily or psychic irritation is followed by reflex general convulsions. In certain organs there is an abnormal reflex excitability of the involuntary muscles: in-



spiratory arrest, asthma and pseudo-croup, enuresis. On account of the importance which the absorption of food assumes at this age nervous disturbances are very liable to arise: nervous anorexia, vomiting. Nervous girls tend to onanism. From the *sixth to the tenth year* the school life begins—so important an event to nervous activity; at this period the frequency of infantile diseases, with their debilitating influences, increases. The neurasthenia now reaches its acme; the emotional and intellectual tension enforces an activity which often leads to physical unrest, sometimes to compulsory movements (*maladie des tics convulsifs*). Even in sleep this unrest continues and occasionally causes night terrors. Enuresis decreases somewhat, onanism becomes more frequent. Some children are unequal to this increased exertion; they become tired and debilitated, headache or migraine appears. In the following lustrum (*eleventh to the fifteenth year*; as a rule, in our observations the last year is not included) there is a certain retrogression of the individual nervous phenomena except that in girls the headache increases—probably in consequence of the deleterious influence of development (chlorosis).

Notwithstanding the fact that in the first years of life the male *sex* is more liable to neurasthenia, a complete summary shows that, if we exclude neuro-hysteria, males and females are affected in nearly equal ratio (in our dispensary service 80 boys, 86 girls).

The *causes of neurasthenia* will be considered in detail in the treatment. Neurasthenic symptoms appear in conjunction with various diseases of the nervous system, and in particular after meningitis, encephalitis, and in degenerates or feeble-minded children; they are also quite usual after chorea and epilepsy. The occurrence of neurasthenic phenomena following exophthalmic goiter (Graves' or Basedow's disease) will be discussed later.

## HYSTERIA

**Hysteria.**—Of special interest and by no means rare, as was explained in the introduction, is the combination of hysteria with neurasthenia, either by the presence of unrecognized hysterical phenomena with the neurasthenia, or by the association of neurasthenic symptoms with well-developed hysteria.

Accordingly, many authors make no distinction between hysteria and neurasthenia and the reports relative to the distribution of hysteria, to the individual *age*, and to *sex*, are widely diverse. Nevertheless most authors, myself included, agree that hysteria is comparatively rare before the seventh year and that from this age females are preëminently affected. My compilations, which cover several years, show that of 64 cases 48 occurred in females and 16 in males: in the fourth and fifth years one boy, 2 girls; from the sixth to the tenth year 9 boys, 11 girls; from the eleventh to the fifteenth year 6 boys, 35 girls; or, with a different arrangement, from the fourth to the eighth year 8 boys, 4 girls; from the ninth to the fifteenth year 8 boys, 45 girls.

This preference for the female sex must not lead to the assumption that

sexual processes of an immediate nature instigate an outbreak of hysteria, although occasionally they may act as psychic traumata; on the contrary, the peculiar physiologic effect of feminine maturity upon the psychic development favors the mediate cause of hysteria, so that, as a rule, the slumbering hereditary predisposition<sup>1</sup> to hysteria becomes manifest at this time. I shall dwell briefly on the nature of this affection, as it does not differ essentially from the hysteria of adults. The psychic character of the disease is acknowledged (I refer to my previous explanation, page 350). There is a peculiar change in the hysterical imagination, in which certain associated ideas, normally of a transient nature, are unconsciously retained and distorted until they give rise to morbid psychic or somatic phenomena. When physical sensations are the object of conception, as generally is the case, they are constantly and unconsciously absorbed in the conception; for example, pain is felt and expressed as abnormally severe, or is still further elaborated by the child in accordance with its lay impression of the human body. Imagination or autosuggestion in children predominates in the motor sphere, less often in the sensory sphere, of the region in question; therefore, upon an ideogenic basis, convulsions and paralysis, over-sensitiveness to pain, or pain paralysis, arise. Besides, the association of ideas which occurs under the wave of consciousness need not always be in excess of this impulse, so that often the patient is not particularly aware of the anesthetics and analgesias. In so far as the associated unconscious chain of perception cannot at all, or only with the greatest difficulty, be severed by conceptions consciously opposed, we speak of hysterical feebleness of will—an expression which may readily give rise to a false interpretation of the psychic mechanism.

The origin of the hysterical manifestations may be widely diverse. An accidental constellation of ideas may produce hysterical phenomena which, under some circumstances, appear in a dream, while the control of consciousness is lost; or an external factor—an exciting discussion, hygienic instruction at school, watching or listening to the complaints of a sick person may produce such a strong corresponding impression that between the reception of the idea and its hysterical development some time will elapse.

Furthermore, by an accidental association of ideas, normal sensation may force an abnormal hysterical conception, or morbid corporeal processes in immediate or mediate sequence may result in a hysterical manifestation; for example, pain may lead at once to a markedly disproportionate reaction—paralysis, syncope, spasms of screaming or other convulsions—or after a disappearance of the actual lesion the corresponding impression of pain, or an associated reaction, may continue or be subsequently revived. In so far as we are concerned with an active accentuation of pain or sensations of exhaustion (*without* other sequels) a differentiation between hysteria and neurasthenia may be

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<sup>1</sup>In the dispensary a nervous predisposition could be determined only in one-third to one-fourth of the cases, but the investigations in this direction do not appear to me to be conclusive.

difficult or even impossible. The mental condition and the psychic habitus must then be invoked as aids in the diagnosis.

The hysterical psychic component may be expected as a regular accompaniment of hysteria even to a less extent in the child than in the adult. Nevertheless, in girls, particularly those of older age, we find the well-known instability and superficiality of the emotions, occasionally a remarkable precocity and loquacity, and a coquetting with events of a personal nature. Their description of their respective troubles is exaggerated and verbose but quite unemotional. Their thoughts are concerned not only with their physical state but also with other matters. Just as with adults will the hysterical child, directly or upon suggestion, tell the most ridiculous or even cunning falsehoods unconcernedly and without the slightest hesitation. The morbid mental condition is indicated—often from earliest childhood—by the hysterical glance. Rarely have I observed hysterical insanity in older girls.

**Head's Zones.**—As regards the character of neurasthenia and hysteria and the relation of the two diseases, the study of *hyperesthesia* is very instructive. In the neurasthenic debility of the nervous system, with stimulation in the course of a nerve segment, there is often a distribution of the irritation within the transverse section, so that pain develops in the corresponding cutaneous area, either spontaneously or by contact. These cutaneous zones, and in particular the conspicuous points of pain, have been closely studied by Head.<sup>1</sup> Quite commonly they are to be found in infancy, in their ordinary distribution, in congenital neurasthenia or in acute neurasthenic conditions (for example, after influenza), and on account of the frequent combination of neurasthenia and hysteria they are also not rare in the latter affection; they may then be expressed in an exaggerated hysterical manner, but, although attention may be diverted from them, as from pain, they cannot be made to disappear by suggestion. "Head's zones" may appear in the neurasthenic subject after the slightest, sometimes physiologic, stimulation. For example, they are not rare in circumscribed areas of the skin over the maturing female breast or over portions of the intestine distended by fecal impaction, and are often demonstrated upon the chest and abdomen without a recognizable cause. Naturally, they are quite as frequent in pathologic stimulation of the organs, for example, over the stomach after it has been irritated by vomiting or otherwise. That these zones appear in hysteria without simultaneous neurasthenia seems to me doubtful. Certainly they should not be regarded as a sign pathognomonic of hysteria, as is still quite usual. The hyperesthesias typical of hysteria, such as hypersensitiveness of the entire cutaneous surface, like the hysterogenic pressure points (ovarialgia), are not common in childhood.

The circumscribed anesthetics and analgesias—particularly upon the extremities in the well-known cuff-like limitation—are also rare. Hypalgesia or analgesia distributed over the entire cutaneous surface or limited to one-

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<sup>1</sup>"Die Sensibilitätsstörungen der Haut bei Visceralerkrankungen." Translation by Wilhelm Seiffer. August Hirschwald, Berlin, 1898.



half of the body (also with involvement of the organs of special sense) is somewhat more common.

Although the stigma of psychic and sensory nature are often disappointing in infantile hysteria, their manifestations are less complex than in the adult. We might therefore speak of a *monosymptomatic* form of hysteria. The diagnostic difficulties are occasionally great, provided there is no suspicion of a nervous affection. Frequently, with the simplicity of the complaint, the foolishness of the underlying conception is an aid to the physician.

Most often we meet with *seizures*: These are often introduced by pain, subjective twitchings and tremors, cardiac palpitation, dyspnea, or flushes of heat. Then, as a rule, with the sensation of *vertigo*, the child is attacked with syncope, in which condition it remains for a varying number of minutes; consciousness usually is more or less retained, but in rare cases is altogether lost. The attacks generally occur in the presence of others—preferably in school, occasionally upon the street—and without injury to the body. From this there are transitions to severe hysterical spasms and convulsions, but rarely, among German children at least, to the hysterical stupor and somnambulism or the great hysterical seizures which have been carefully studied by the Charcot school.

Naturally, it is impossible to discuss all of the various expressions of the hysterical imagination, but some of the more common forms must be briefly mentioned. Often there is a complaint of twitching in various parts of the body at a definite or indefinite time, or from some particular cause. From these, in older children, tics or localized spasms arise. Occasionally one or another extremity becomes rigid or performs apparently involuntary, definite movements, or, in consequence of unconscious, more or less close imitation, some pathologic phenomena are presented. Even young children not infrequently declare that a certain portion of their body, in which, transitorily, there has been an unpleasant or painful sensation, cannot be moved; therefore, that they cannot rotate the head, cannot sit erect, cannot stand nor walk (*astasia abasia*). While the recognition of this pseudoparalysis is not difficult, the hysterical pain which continues in the youthful imagination after a fleeting period of reality, and produces a corresponding imitation of movement, atrophy, etc., may not be properly appreciated for a long time.

No less common, and under some circumstances difficult to estimate, are the *pains* which simulate disease of the internal organs and are due to a memory of former pain or to an active imitative sensation. In children these sensations are mostly in the head and stomach.

Besides the vertigo and pain phenomena, quite often there are disturbances in the *respiratory, digestive, and urinary passages*. Symptoms relating to the special senses, and vasomotor and secretory disturbances, are rare, at least in my experience. Thus, we meet with cough, hoarseness, sobbing, and especially asthmatic complaints of hysterical nature in which autosuggestion or unconscious imitation plays a part. Further, there is retching, eructation, vomiting, inability to eat or digest certain foods, and at last a general disinclination for

nourishment. To avoid confusion I shall describe *hysterical vomiting*, which is so common, and contrast it with the also common neurasthenic vomiting. The latter condition is an easily induced reflex which cannot be suppressed on account of nervous weakness and is due to the eating or thought of something obnoxious. In the former condition the subconscious memory of an act of vomiting, which was observed in another person or performed by the hysterical child itself, is afterwards translated into the corresponding muscular action by an abnormally vivid conception image, therefore upon attempts to partake of food. Thus, a child, whose grandmother suffered from emesis due to carcinoma of the stomach, vomited after any excitement; another child who first vomited after a dose of medicine, subsequently repeated the act after each attempt to take food. Under some circumstances, however, neurasthenic vomiting cannot be differentiated from the hysterical form.

With *difficult defecation* the circumstances are quite similar: Through an insufficient control of the will *habit constipation* often develops, either in consequence of a weakening of the spontaneous stimulation of evacuation by frequent artificial aid, or because the nervously active and flighty child does not give sufficient attention and effort to the stimulus which arises. In the former case, as Thiemich<sup>1</sup> rightly emphasizes, a cure can be accomplished by the withdrawal of all treatment, but I hesitate, in the one case as in the other, to deduce a diagnosis of hysteria from the successful stimulation of the will. On the other hand, it is not to be denied that the hysterical idea of not being able to defecate can lead to habitual constipation. I saw hysterical constipation, as a consequence of disgust, in two young boys. "It is dreadful to have a movement of the bowels" was the statement of a boy aged three who never had a desire to defecate when reminded of it, except at night, at which time the inhibitive conception was less strong and he therefore called when there was an inclination for stool. During the day, instead of the perpetual constipation observed in another case, he voided feces and urine into his trousers.

*Involuntary passage of urine* may be either of a neurasthenic or hysterical nature. I assume the latter to be the case if, as already mentioned, at an inauspicious moment there is an irresistible impulse to urinate and the conception is at once translated into the act.

As little as it is possible generally to construe all of the modes of manifestation of hysteria in advance, just as little is this possible in infantile hysteria. Not only are functional diseases simulated in hysteria, but under some circumstances, through a hysterical disturbance of innervation, they may actually exist. For example, it is presumed that the stomach is not capable of digesting mild food, whereas the immoderate introduction of all kinds of food, and even that difficult to digest, shows that the organ can functionate normally. Certain spastic conditions of the muscles of the stomach, which lead to gastrectasis and its sequels, possibly belong to this category.

**Diagnosis.**—In the *diagnosis of functional neurosis* (as always in the exam-

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<sup>1</sup> *Jahrbuch für Kinderheilkunde*, N. F., VIII, p. 886.

ination of children), it is primarily advisable to lead the patient into conversation. This permits an insight into the mental condition. The behavior of the child, its emotions and intelligence, and especially the nature of its complaints and the manner in which they are described, as a rule will readily indicate that we are not concerned with a pure somatic disease; hence we investigate the nervous phenomena, of which there is no special complaint. Associated with this is the physical examination, the importance of which in infancy cannot be emphasized too greatly, since confusion with organic diseases—even of the nervous system—may readily occur. All of the aids of diagnosis—ophthalmoscope, X-rays, etc.—are to be invoked. The beginning of a brain tumor, cardiac insufficiency, albuminuria, or neuphritis may readily be overlooked! On the other hand, it is scarcely necessary to mention that a functional neurosis may have for its sequel an organic disease; for example, nocturnal enuresis, an inflammation of the bladder; neurasthenic debility, brought about perhaps by onanism, an insufficient activity of the heart. Here again we must mention that a substantive organic affection (as an injury) may appear under the guise of a neurosis. If it has been determined conclusively that the child is suffering from a neurosis, it is important for the diagnosis, prognosis, and treatment to understand clearly the importance of heredity, social and educational factors, and, for the treatment of the individual symptoms, the opportunity for psychic contagion. Finally, the investigation of the case must lead to a differentiation between hysteria and neurasthenia. Not infrequently the diagnosis only becomes positive after the success of the treatment has been manifested.

## TREATMENT OF NEURASTHENIA AND HYSTERIA

As the treatment of neurasthenia and of hysteria has much in common, and there are but few factors in opposition, to save repetition these two affections will be considered together. While the abnormal predisposition cannot be removed, and the symptoms dependent upon it frequently cannot be dispelled for internal or accidental external reasons, nevertheless our therapeutic efforts, in these nervous affections which may seriously and permanently implicate the welfare of the patient and his enjoyment of life, are not to be undervalued, since it is most easy to attack the root of the evil in infancy. First we shall concern ourselves with the neurasthenic and hysterical conceptions, and afterwards with the individual symptoms to which they give rise. Rarely in hysteria, but more commonly in neurohysteria and in pure neurasthenia, the general condition of health is below the normal, and its improvement is a prerequisite in the cure of the nervous affection. In so far as we are concerned with an acute cause in the outbreak of neurasthenia, the diseases of infancy are operative among the somatic factors, and perhaps quite as frequently acute catarrh of the respiratory passages, which is mostly the result of influenza. Among the chronic diseases which add fuel to the neurasthenic complaint are the prolonged dyspepsias which occur during the first years; next in frequency



are scrofula and tuberculosis, and toward puberty (in girls) chlorosis. It must be mentioned that the so-called anemia—the frequent accompaniment of neurasthenia—seldom depends upon a decrease of hemoglobin, but is usually due to an enervation of the cardiac activity and to unfavorable vasomotor stimulation of the vessels. If preparations of iron are efficacious in this condition their mode of action must be quite different from the ordinary.

I shall not enter in detail into the therapy of organic affections but will pass on to the *hygienic and dietetic treatment* of neurasthenics who are not organically diseased, and which is also of value in hysteria. In growing children the nutrition must be abundant, provided there is no tendency to obesity. The administration of meat broths is to be limited or entirely avoided; only in cases of chronic gastric dyspepsia are they indicated. Meat should be given but once daily, and in nervous children should be eliminated entirely in the first few years. One or at most two eggs are permissible, provided they are well borne. A vegetable diet, with a plentiful addition of milk and butter, is essential. Instead of spices and sweets the child should be given fruit. Coffee and alcohol are to be forbidden. Cocoa and chocolate must be given in moderation, as even they may be stimulating. The meals (about five per day) are to be evenly distributed, and the last should not be too close to bed-time. They are not to be too abundant nor rich in water.

The clothing should be simple and light, and suited to the season. Coddling is to be avoided, as well as the incomprehensible method of inurement. The head should be free; the limbs, which are apt to be cool and livid, must be kept warm. For children who sweat readily underclothing of cotton or wool instead of linen is advisable. Furs and starched undergarments should be avoided. The neck must not be constricted nor the trousers too narrow. The child should have fresh, cool air even at night (60°-65° F.) and should be out of doors as much as possible, concern itself with play, mild sport, or light work, and not be permitted to become low-spirited. It is better to avoid a constant caution against taking cold, injury, and the like. Should there be slight accidents, as a fall, a cut, etc., not much ado should be made of it. The resistance of the skin and the power of the muscles must be strengthened. For poorer school-children, some auxiliary occupation in the open air is more serviceable than sitting upon the benches of charitable institutions or in their gloomy homes. In how far a residence at the seashore or in the mountains will be beneficial to the nervous but otherwise healthy child cannot always be determined in advance. A residence at the seacoast where there is a decided tide is often too exciting and gives rise to headache and restless sleep, enuresis, or after their return home, a relapse of bronchial asthma. Sleep should be abundant and should be watched on account of the possibility of onanism. Even older nervous children should rest for two hours after the mid-day meal. "Early to bed and early to rise!" It is well known how often nervous patients combat this advice.

In hydrotherapy we must individualize, as nervous persons react quite differently to its employment. Water improperly employed may produce a ner-

vous condition; with continued and monotonous use its effect is exhausted, with slight periodic interruptions it invigorates and hardens. Too great cold, too prolonged employment of an individual method, over-strong mechanical stimulation (by the douche, by friction) must be avoided. Luke-warm half-baths, mild ablutions, moderate friction, brief lukewarm douches, and, with great unrest and insomnia, prolonged warm baths, are indicated in the individual case.

An essential feature in the control of the functional neuroses which concern us here is *psychic treatment*. The irritable nervous weakness which we meet with, alone or in combination with hysteria, is usually not amenable to cure; it may, however, be seriously increased by unsuitable stimulation above the given minimum. Even immoderate bodily exertion will exhaust the nerves, and much more so the combination of physical and mental exertion such as is brought about by attending school. If at the onset a certain amount of exhaustion is present in healthy children (in addition to arrest or decrease in weight), in those of a nervous predisposition, as we have seen, quite a horde of neurasthenic affections arises or becomes aggravated. Only the over-burdening of the mind by the instruction of the higher schools will be suggested here. Sickly children and those of feeble mentality—neurasthenics are often both—succumb in a marked degree to the injurious effects of school instruction. Here, and naturally also in private teaching, there must be an individualization according to the powers of tension and conception. The company of other children, as is brought about by attendance at school, usually serves to counteract a dangerous egotism, anxious self-observation, physical and psychic effeminacy and prematurity, which might result from association with adults. In any event it is well to see to it that children who are mentally deficient are not injured by their schoolmates, and that those who are of a hysterical temperament are not exposed to psychic contagion.

But even prior, and in addition, to school influences other deleterious agents have an effect upon the nervous child. Here, since the importance of disease has already been emphasized, only bodily and psychic trauma will be mentioned: Fright of all kinds, injury, sometimes the slightest but at the same time unavoidable, sexual abuse, exciting experiences in the street, all give rise to neurasthenic or hysterical symptoms; therefore such influences should be avoided in so far as possible. I need only suggest the danger of children sleeping together, of intercourse with depraved playmates, visits to the circus, museums, theaters, the reading of newspapers, Indian stories, blood-curdling novels, etc.

Most important in the treatment of infantile neurosis is the *influence of the parents*, in so far as they are an example and the companions of the child, and prescribe and carry out their education. If they themselves show more or less conspicuous nervous disturbances, corresponding conceptions are awakened in the child which stimulate the hysterical imagination in a definite direction. Abnormalities in character, in the emotional temperament, in the sphere of the will, may produce great educational injury. In the neuropathically

predisposed child the results of parental alcoholism, poverty, or the death of a parent, which arise from harshness or over-indulgence, are especially early and severe.

*Education*—in the neurosis under consideration—in hysteria and neurasthenia in various strength and direction—may be preventive or may moderate the course and facilitate the psychic treatment of the individual symptoms. Aside from the training by the formation of proper conceptions and theories, the education should lead to a normal development of the emotions and of the will. Further, it should be mentioned that, as in the development of the motor so also in that of the psychic activity, the development of the inhibitive property is of importance. As the education aims in this direction in the nursling in the absorption of food and the discharge of the excreta, so is its tendency later in a more extensive degree; therefore the ingrafting of the inhibitive faculty is of especial value in the treatment of neurasthenia and of hysteria. When the physician enforces the educational requirements he must expect that they will develop and facilitate the normal activity of the organs by the constant alternation of exercise and rest, that they will develop the will to prompt and conscious action, and that the intellectual interests will expand in an altruistic direction. Therefore it is necessary to combat the consideration of bodily conditions, in so far as this is superfluous or harmful to health, and to prevent a premature development of conceptions and theories which will interfere with the proper exercise of thoughts and actions suited to the age of the child, and make it precocious and inquisitive, thereby annihilating the proper educational influence. Simplicity, clearness, a proper sequence in the psychic development, self-sufficiency, and a hardening of the body should be strived for. If the more or less abnormal parents cannot direct the education into these channels the child should, if possible, be removed from its environment. Naturally, it is often enough difficult to find conditions which permit of a proper training. This is more practicable with persons of the middle class in the country or in small towns.

Well-trained children, i. e., those who can be guided by proper influences, may occasionally suppress sensations and reflexes (cough, vomiting) due to an organic disease and thereby facilitate its disappearance. How much more may this be possible when the symptoms have their essential basis in a functional defect of the nervous system. If, therefore, we can successfully enlist the conscious will of the patient in the *treatment of individual symptoms*, this aid is all the more necessary, especially in hysteria, to influence in a mediate manner the thoughts of the patient and the actions which arise therefrom. Conceited, flighty, and inattentive children are exceedingly difficult to influence by any form of treatment.

Indirect guidance of the will results from *suggestion*; it dispenses with the objective, true, and logical explanation and contents itself with a subjective proof which is evident to the patient. In rare cases only hypnosis succeeds in directing the unconscious thoughts into the channel intended; in most cases its accomplishment is possible by suggestion when the child is awake. For this



it is necessary for the physician to have the entire confidence of the patient, as otherwise his statements would not be credited and would not be translated into instinctive conclusions; therefore the hysterical patient must not be mortified by a wrong reproach of a deliberate deception, nor must the reprimand be uttered by those about him. The friends and relatives had better remain ignorant as to the nature of the disease, as they will rarely understand the difference between deception and imagination, particularly when, as in hysteria, the one is occasionally added to the other.

Chastisement at the proper time may occasionally, especially in young children, stop the tendency to vomit; retiring to bed late prevents nocturnal enuresis and similar symptoms. The association of a disagreeable sensation with the process in question acts upon the imagination—notwithstanding the fact that these processes appear to be more or less remote from voluntary control of the will.

As a rule, besides the actual suggestion certain logical processes of thought, no matter how naïve, which act involuntarily, must be associated with the idea of repugnance: Thus drugs of a disagreeable taste, the electric current, or other unpleasant therapeutic agents do not act directly by the disagreeable sensation produced by them, but by the idea that they have a particular efficacy. A firm impression of this idea is usually serviceable: I take the child's head firmly between my hands and while he looks directly at me I utter distinctly the context of the conception and then have my words repeated by him.

But the conviction of the curability, and simultaneously with this the cure, may be attained by other methods, for example, by gradually progressive exercises. The best exercises are those of a rhythmic nature which involve the corresponding muscles upon the normal side. They may also take the form of an interesting amusement in which the play of the diseased and healthy muscles respectively is very difficult to distinguish (for example, piano playing). Or we may proceed by the method of irruption ("Ueberrumpfung," Bruns), in that the patient is surprised or frightened into producing involuntary movements with the hysterically paralyzed muscles. By this means he will perceive the capability of movement and thus regain the power. These methods are various. Occasionally an energetic command is sufficient to arrest hiccough, crying, vomiting, and the like. However, I have more frequently seen relapses follow this method. It must be remarked that the unconscious processes of sleep (night terrors, automatic movements, somnambulistic conditions) are also susceptible to a certain degree to the suggestion of the wakeful period. If this method fails hypnotism may be tried.

In contrast to active suggestion in neurasthenic and hysterical conditions is their simple neglect: The patient is treated by ignoring the symptom of which he complains. While he adheres to this symptom because instinctively a pleasant conception is associated with it (pity, admiration), by this procedure the patient's interest in his trouble is lessened and gradually other ideas become more prominent. To institute this method successfully a removal from the usual conditions is necessary; therefore hospital treatment is indicated.

If the treatment of pure hysteria has been successful it is necessary to avoid any future reference to the symptoms. As a matter of fact, sequels which require further consideration are rare; for example, atrophy after prolonged hysterical paralysis, emaciation after tenacious vomiting. In neurasthenia the conditions are different! When psychic treatment has produced a cure, the function of the organ in question has been debilitated from the onset and remains unstable even after recovery, so that there is every reason to strengthen, or at least to protect, the organ from debilitating influences and to continue the treatment after cure. Nevertheless, in neurohysteria particularly, we must skillfully avoid supplying fuel to the hysterical imagination.

I must mention a few measures which are serviceable in this sense in the treatment of some of the symptoms of infantile neurasthenia. A complete description is not my object, for they have in part merely a suggestive effect and therefore may be varied according to discretion; on the other hand it is not necessary to detail repeatedly the necessity of a general strengthening of the nervous system.

In the *treatment of headache* and of *typical migraine* mental rest and plentiful fresh air are essential. All hindrance of the circulation by clothing or by a sitting or stooping posture is to be prevented. Massage of the nape of the neck sometimes has a favorable effect, but general massage may—according to the vasomotor conditions present—occasionally produce the attack. Headache and nausea upon awakening, with pallor of the face, are ameliorated by a warm breakfast before rising, or by bathing with hot water, particularly of the face. In angiospastic headache a good symptomatic effect is produced by nitroglycerin (a one per cent. alcoholic solution in doses of one to three drops, or 1 to 1.5 milligrams in tablet), to be taken for the attack or as a prophylactic. Applications to the head, or binding it with a silk cloth, or the employment of the menthol pencil or of electricity in its various forms may be mentioned. For all varieties of headache doses of antipyrin, citrophen, phenacetin, etc., may be given according to the age, as in adults.

The prolonged use of quinin is very serviceable for the control of headache and in fact all forms of nervous debility (0.03-0.1 three times daily, but not upon an empty stomach). Often the administration of compound tincture of cinchona, aromatic tincture, or the ethereal tincture of valerian (of the first, 10 drops to a teaspoonful in sweetened water three times daily, to be taken a few minutes before meals; of the last two tinctures, smaller doses of 10 to 15 drops, given in the same manner) are sufficient. In addition to these the cautious employment of hydrotherapy!

For the prevention of *pseudocroup* and of *bronchial asthma*, the therapy of which is not to be discussed here, acute inflammation of the respiratory passages and especially all infectious catarrhs are to be avoided. After an infection has once arisen new infections readily occur, particularly in the cold months, and cannot positively be eluded even by a residence in a mild climate. After complete cure of the catarrh, never earlier, a hardening method directed against the changes in temperature is to be attempted. The nervous components of

asthma may be decreased or even entirely overcome by a proper hydrotherapy of five or six weeks' duration, or by treatment with arsenic for two or three months. Residence at the seashore is not usually beneficial, but a high altitude is liable to have a favorable influence on the nervous system.

Very frequently the enlarged pharyngeal tonsil has been removed to relieve the tendency to asthma, but always without the desired success. While the reflex irritation of the bronchial tree from the nose, without producing a particularly frequent tendency to asthma, cannot be denied, the pharyngeal tonsil is included in this category only because of the clinical exaggeration which its enlargement at present commands. There is rather a certain relation between adenoid vegetations and the nervous disturbances of sleep which are produced by them through an obstruction of the nasal respiration, such as restlessness, unpleasant dreams, perhaps also night terrors and enuresis. But even if this is admitted, we are dealing only with an accidental cause in children who are already neurasthenic. Besides, if a favorable result should follow the removal of a tonsil in the individual case a suggestive influence of the operation must be considered.

To prevent the *nocturnal disturbances* the following measures are of value: A light and not too plentiful evening meal deficient in water (therefore gruel or a sandwich, followed by fruit); retiring to bed at the earliest an hour after the supper. In the interval lessons, exciting games, reading and entertainment are to be denied. Before going to sleep the natural necessities are to be satisfied, the bedroom is to be cooled, well-ventilated and quiet, and the bed not too soft nor too warm. The child should fall asleep quickly—if necessary by the aid of command or suggestion. The hands are to be kept over the bedclothes and the child is not to lie upon the abdomen.

Whether in addition to a hindrance of nasal respiration and overloading of the stomach, the irritation caused by worms is capable of producing these nocturnal disturbances (as is reported) is unknown to me. It is certain that any exciting experience during the day may influence the sleep of the nervous child and cause disquiet. If, after removal of the physical causes which may be present, the disturbance of sleep continues, suggestion may occasionally be employed with advantage. Among drugs, valerian (cold valerian tea given before bedtime) or the bromids may be considered. I shall not discuss the irritative conditions which arise during the night in consequence of febrile diseases.

A few words must be devoted to *nocturnal enuresis*. Apart from the psychic treatment—punishment, the electric current and other remedies that have a suggestive action—repeated rousing of the patient may prevent an over-filling of the bladder; or by raising the lower portion of the bed the pressure of the urine upon the constrictor muscle of the bladder (?) may be lessened. If we consider nocturnal enuresis in association with diurnal enuresis and with pollakiuria, among the mechanical remedies the distension of the bladder by the injection of an antiseptic solution (3 per cent, boric acid)—with sufficient rinsing to avoid cystitis—has often been useful. On the other hand, a suggestive



effect is occasionally produced by the distension of the constrictor muscle by a large catheter.<sup>1</sup>

Among drugs the fluid extract of *rhus aromatica* (10 to 15 drops three times daily) has shown some effect, at least so long as it is administered. The employment of water in the form of lukewarm Sitz baths or brief half-baths is useful (two to three minutes at a temperature of 30° C.—86° F.). [*Belladonna* in a sufficient dose at bed time will relieve the irritability of the detrusor. If the sphincter happens to be so weak as to allow incontinence in day time, strychnia with or without ergot, also the interrupted electric current, will strengthen it. But do not tamper with drugs; give an effective dose or leave them alone.—EDITOR.]

Finally, a few words regarding the treatment of *masturbation*. The cardinal indication is to make the diagnosis. In no instance must mechanical appliances come in contact with the genitalia; therefore all rings, covers, and the like are to be rejected. Children in the first two years of life, who masturbate in bed, are to be lifted. According to the manner of manipulation of the hands the sleeves are to be crossed or fastened to the side of the bed, the lower opening in the shirt is to be sewed, or the hands are to be kept away from the sexual organs by a sack-like covering. If the legs are pressed together they are to be crossed and tied, or firm, straight sockets which are held apart by a transverse staff, are attached to the knees and partially envelop the upper and lower leg. In one case I successfully employed a board which was placed lengthwise between the legs and passed through an opening in another board placed horizontally across the bed. Older children may wear a leather bandage during the night similar to swimming tights, with openings on the sides; the front portion extends upward over the umbilicus; as in old armor, there is sufficient space in the region of the genitalia to avoid contact of the erect member with the bandage. Such mechanical measures are only to be introduced when close watching of the patient is impossible, and should be employed uninterruptedly for several years.<sup>2</sup> In cases in which onanism is practised only under certain circumstances the treatment is simplified. While the impulse for onanism is so strong in severely neuropathic children that moral instruction and logic are likely to be unsuccessful, in those cases which are to be regarded more as vicious habit education is apt to be successful. Among drugs, the occasional employment of the salts of bromin must be considered. Too great stimulation and irritation by hydrotherapy should be avoided. Tiring occupation in the open air, as riding, bicycling, swimming, etc., divert attention from the sexual sphere and are therefore useful; but it is necessary to note that these exercises may produce erection if there be great irritability, and they are too strenuous where there is great exhaustion. [Camphor and lupulin should be tried; camphor monobromate is easily taken.—EDITOR.]

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<sup>1</sup> The inelastic male catheter of low number is unsuitable for catheterization of boys.

<sup>2</sup> Special attention should be devoted to the child while on the toilet.

## PROGNOSIS

In a summary of the treatment reference must be made to the prognosis. In hysterical as well as neurasthenic children removal from the accustomed environment into surroundings which are more advantageous to bodily and mental development is very beneficial, but usually the circumstances are such as to prevent a recourse to this aid. Occasionally adolescence itself brings about such a favorable change as to obscure the neuropathic condition for the time. In other cases, particularly in the hysteria of females, the disease may now assume a severe form. In general the symptoms of hysteria, which are usually monosymptomatic, and with them the symptoms of hysteria in childhood, may be readily alleviated. In neurasthenia, as well as in its combination with hysteria, a careful consideration of the individual nervous symptoms, particularly as they aggravate the general nervous condition, is advisable. Here, under some circumstances, we may interfere successfully in definite directions. With this, in every case, the general treatment according to hygienic-dietetic laws must be considered, in which therapeutic measures, at first physical, later by drugs (for example, the bromids), is to be included. Neurasthenia may be improved or cured the more readily if it appears somewhat acutely from some definite cause. The treatment is more difficult if the condition occurs in combination with organic disease (as scrofula), or with nervous affections (as Graves' disease, epilepsy, etc.). The earlier and more severe the neuropathic hereditary degeneration is manifest the less may we count upon an improvement, to say nothing of a disappearance, of the nervous phenomena.

## ORGANIC AFFECTIONS

Following neurasthenia and hysteria, and their subdivisions, a second group of diseases must be considered, which represents a transition from the purely functional to the organic affections of the nervous system: Among these are chorea minor (St. Vitus' dance), Graves' disease (exophthalmic goiter), and epilepsy.

I need not enter into the consideration of **chorea**, as a special article in this volume is devoted to it.<sup>1</sup> I shall only mention that I believe its relation to acute rheumatic fever, in a pathogenetic respect, is very intimate and therefore I would include it in this group of maladies. In favor of the rheumatic nature of chorea is its frequency in rheumatic families—that is, in individuals rheumatically predisposed—its coincidence with other rheumatic affections, its increase at particular seasons, and the like.<sup>2</sup>

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<sup>1</sup> B. Bendix, Chorea Minor, this volume.

<sup>2</sup> Compare Erwin Kobrak, "Ueber rheumatische Chorea und ihre anti-rheumatische Therapie." *Arch. f. Kinderhk.*, Bd. XXVI, Heft 1-2.

**Graves' Disease.**—The position of Graves' disease among the neuroses is indefinite. Having learned to recognize the phenomena of an increase of function of the thyroid gland through feeding it is not too much to assume that some of the symptoms are in relation with an enlargement of the thyroid gland. Upon the other hand, however, those persons are frequently attacked who are of a neuropathic predisposition or simultaneously present other nervous diseases which are not in causal connection with exophthalmic goiter (Basedow's disease).

As the enlargement of the thyroid gland in Graves' disease, likewise as in simple parenchymatous goiter, is in marked dependence upon the development of the female sexual organs, although in what way is still unknown, exophthalmic goiter is rarely observed in infancy, and, similarly to hysteria, we find the affection distributed according to sex and age; for example, among 37 cases only 9 occurred in males (3 before the sixth and tenth years and 6 before the eleventh and fourteenth years); 28 among females (5 from the eighth to the tenth year, 24 from the eleventh to the fifteenth year). That Graves' disease itself, or goiter or exophthalmus, occurs in the ascendants or among brothers and sisters simultaneously is well known (4 times in my cases). Very severe types of the disease are exceptional in infancy. The individual symptoms are not marked—the goiter probably is most prominent—and are rarely fully developed at the same time. Thus in the 37 cases above mentioned goiter was absent 6 times, exophthalmus 8 times, tachycardia 11 times; tremor was present in only 8 cases. With the exophthalmus (29 times), which was often very slight, a more or less distinct Graefe's sign (failure of the upper lid to follow the eyeball in glancing downward) was associated in 26 instances, only 7 times Stellwag's symptom (apparent widening of the palpebral aperture), and only 3 times Möbius's sign (deficient convergence). In spite of common tachycardia (26 times), cardiac palpitation was rarely (12 times) complained of. Ascending flushes and marked sweating were rare (in 7 and 4 cases respectively). The classical triad—tachycardia, goiter, exophthalmus—was present only 14 times in 37 cases; 5 times the goiter was absent, 7 times the exophthalmus, and 9 times tachycardia, the other two symptoms of the triad occurring together. In two cases there were exophthalmus and goiter; in one of these, however—besides Graefe's symptom—there were tremor, sweating and flushing; in the other case the diagnosis was rendered positive by a simultaneous tremor. In contrast to the 37 positive cases of Graves' disease there were 12<sup>1</sup> in which the combination of symptoms did not justify a positive diagnosis.

Headache of varying intensity is very common in Graves' disease (22 times in the 37 cases). This as well as the slight irritability, may be regarded as a symptom of the disease, but on the other hand, as is indicated by the common hereditary nervous predisposition (9 in 36), may be considered a nervous dis-

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<sup>1</sup> Of these 12 children, 10 were females, and of these again 7 were between 11 and 14 years of age.



turbance which is commonly combined with Graves' disease. In these 37 cases the various symptoms of neurasthenia were present 13 times; once there was conspicuous *maladie des tics*, once stuttering, and 5 times hysterical phenomena. Of these 37 children 7 were of feeble mentality.

*Treatment.*—The frequency with which the symptoms of irritable weakness become prominent indicates that the general measures which were advised in the treatment of neurasthenia are to be employed here. The cautious use of hydrotherapy is particularly advantageous; internally arsenic—in association with iron, in the form of Levico water, a teaspoonful three times daily—given in courses of six weeks. When the symptoms are prominent removal from school and a residence in the country or in a moderately mountainous region are desirable. In regard to the specific treatment of exophthalmic goiter with serum, thus far nothing positive can be stated. Galvanism of the cervical sympathetic may always be employed in cases of marked goiter and tachycardia. Cardiac palpitation may require local, cold compresses.<sup>1</sup>

**Epilepsy.**—The third disease which is on the border-line between the functional disturbances of the nervous system and organic affections is epilepsy. Its occurrence in infancy is of great interest, if only for the reason that the heredity and the etiology may be discerned during this period with greater certainty than later. The differentiation of the epileptic attack from other convulsions of early childhood is naturally difficult. The separation from the great class of convulsions which arise from tetanoid hyperirritability has only lately been possible, and the differentiation from febrile convulsions and from that form which I designated previously as apneic arrest of breathing has not been made sufficiently clear. To this must be added the fact that, although in very rare cases, children who have first suffered from one of the above mentioned forms of convulsion have later developed typical epileptic attacks. While, under some circumstances, one variety of convulsion may emerge from another, in other cases, according to my positive experience, there is an accidental condition (for example, an infant suffering from convulsions due to cerebral syphilis: cured; later tetany: cured).

Even if those not uncommon cases are left entirely out of consideration in which the observation of the small child yields no positive certainty regarding the nature of the convulsive attack, and if we adhere essentially to those cases which retrospectively have been rendered certain, it is shown that epilepsy generally begins in the first five years of life—to be exact, from the second to the fifth year—less commonly between the sixth and tenth years, and is somewhat rare from then to the fourteenth year. The male sex, perhaps, is more greatly implicated in the first years; a preponderance in females about the time of sexual maturity does not occur in epilepsy, as was observed in hysteria, chorea, and Graves' disease.

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<sup>1</sup> See also volume on "Diseases of the Nervous System," p. 960, *et seq.*

Accurate reports are contained in the following table:

AGE	BEGINNING OF TYPICAL ATTACK			BEGINNING OF TREATMENT		
	Males	Females	Total	Males	Females	Total
Under 1 year.....	4	1	5	..	..	..
1 year .....	11	5	16	5	2	7
2 years .....	7	4	11	5	2	7
3 " .....	4	5	9	3	3	6
4 " .....	3	4	7	5	2	7
To end of 4th year	29	19	48	18	9	27
5 years .....	3	2	5	3	2	5
6 " .....	1	5	6	5	2	7
7 " .....	3	3	6	2	2	4
8 " .....	2	2	4	3	4	7
9 " .....	5	6	11	6	7	13
5th to 9th year	14	18	32	19	17	36
10 years .....	2	..	2	5	7	12
11 " .....	3	3	6	4	5	9
12 " .....	3	2	5	6	6	12
13 " .....	1	..	1	2	3	5
14 " .....	..	1	1	..	1	1
10th to 14th year	9	6	15	17	22	39
Onset unknown	2	5	7	..	..	..
Total	54	48	102	54	48	102

To a certain extent the reason that epilepsy occurs in early infancy is that at this time diseases of the brain and of its membranes, and in part also severe injuries to the head are most apt to occur, which later give rise to convulsions, especially encephalitis. But in spite of the fact that our anamnesis represents the greatest possible accuracy, there were among 102 cases of genuine epilepsy—Jacksonian epilepsy is not considered here—only 11 cases of this kind. An inflammation of the brain or of its membranes was noted 3 times in the first year, once in the fifth year, 2 times in the seventh year. An injury to the head which could be regarded as responsible for epilepsy, once in the second year, once in the fourth year, 2 times in the fifth year, once in the tenth year. In addition, in 9 cases organic disease existed prior to the outbreak of the convulsions, which were *perhaps* in causal connection: Injury to the head 3 times, 6 times severe affections with prolonged unconsciousness or "tonic spasm" (among these once enteric fever, once scarlatina and diphtheria, once measles).<sup>1</sup> That organic changes might also be demonstrated in other cases, although perhaps only of a microscopic nature, can scarcely be doubted: This is sometimes indicated by the accompanying lack of mental development which naturally must not be included with weakened intelligence

<sup>1</sup> In these 9 cases there were only 3 of a neuropathic predisposition in so far as the mother was nervous or suffered from migraine.

or memory, and which may appear as a *consequence* of the convulsive attack. Among 52 children 42 were of inferior mental capacity; of these 12 were actual imbeciles.

While the convulsions, the origin of which may be referred to acute diseases of the brain, may appear independently of any hereditary predisposition, genuine epilepsy in the strict sense of the term is usually recognized as a degenerative neurosis. Among nervous diseases in the ascendants, in the 91 of my cases which remain after deducting the 11 due to organic disease, the following was shown: the father or mother or both were nervous or suffered from headache or migraine in 16 cases (19 per cent.); once the grandmother suffered from head colic, once the grandfather died of a mental disease; in the father or in the mother, or in their families, convulsions were noted 5 times (5 per cent.); in 3 cases the father suffered from lead intoxication, and in 8 cases he was addicted to alcohol (9 per cent.); also one case in which the mother was nervous.<sup>1</sup> Notwithstanding the fact that this small compilation gives sufficient support for the importance of a neuropathic predisposition (37 per cent.) too great value should not be attached to it individually. For example, the importance of alcoholism, although usually great stress is laid upon it, cannot be readily estimated, as other causal factors may be associated.

Thus, in one of my cases the dipsomaniac father, who later developed hydrocephalus, gave copious alcoholic drinks to both of his sons prior to the appearance of their convulsive attacks. The significance of syphilis in epilepsy is difficult to estimate. Even when the cases due to cerebral syphilis (with their usually cumulative attacks) are left out of consideration it is still conceivable that under certain circumstances the disease may be of a parasymphilitic nature. It is noteworthy, however, that in none of my cases of genuine epilepsy, in which, in addition to the patient the brothers and sisters were well known to me, could syphilis be definitely demonstrated.

Although all forms of epilepsy are found in infancy, nevertheless their manner of appearance is influenced by the age and, so to speak, by the youth of the disease.

Frequently epilepsy is seen in its mildest forms. In rare cases even loss of consciousness is absent at the onset. Thus, one of my patients who suffered from scarlet fever complicated by disease of the brain, at the age of 6½ years had at first as a sequel attacks of pain and pricking in the right side of the head, with spasm in the forehead and right eye but without loss of consciousness, and not until the 13th year did severe typical attacks of convulsions develop, and, in addition, attacks of *petit mal* as well as psychic equivalents with perambulation (objectively, right-sided hemianopsia). Very frequently (31 times in 84 cases) epilepsy was present in the mildest form. Occasionally there was momentary unconsciousness: A transitory vertigo or loss of consciousness, with headache and nausea appeared, or the child had a fleeting rigid expression, made a grasping motion or slight contraction, or a rapid rota-

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<sup>1</sup> In 3 cases the labor was severe (twice with forceps).



tion of the eyes. Frequently also, in these mild attacks (*petit mal*), there are convulsions of brief duration which are of a tonic nature, not at all or only to the slightest extent clonic and therefore are not considered as such: The child becomes pale, his head drops backward, or, as is more usual, he falls, remains prone for a few seconds or minutes with loss of consciousness, and the attack is over. In these milder cases sleep does not always follow, as is the rule in the severe forms, and an aura may precede the attack: the child is suddenly frightened, screams, clutches tightly (a child of one year), or runs around as though intoxicated, crying "I fall," and has a simultaneous nausea (child aged three). Sometimes the headache, nausea, and vomiting follow instead of precede the attack. Among the premonitory auræ the sensation of pain in the abdomen must be mentioned, as well as formication.

Occasionally these mild attacks appear at the beginning of the epileptic disease; but often they occur in irregular alternation with severe convulsions and psychic disturbances. Among 84 cases different manifestations appeared in the same patient in 23 instances. The great typical attacks require no description. Not rarely—10 times among 84 cases—they occurred only or usually at night, either after falling asleep, in the course of the night, or upon awakening. In rare instances the attacks betrayed themselves by the involuntary evacuation of urine. The initial cry and biting of the tongue are more rare in children than in adults, and psychic disturbances as a rule are prominent: During or after the attack the child may be silly, ill-natured, and confused.

In rare cases the number and the severity of the attacks increase and we then have a *status epilepticus*. Here, naturally, a permanent damage occurs to the mental activity, whereas otherwise it is of irregular frequency and severity.

Unfortunately it is impossible to state that the early institution of a treatment with bromids renders the *prognosis* of epilepsy more favorable. Although the frequency of the attacks and with this the nerve-racking effect is decreased, the probability of final cure does not become greater. With a diet in which salt is eliminated as much as possible a large dose of some bromid (0.5—2 grammes) in milk is given at night—when there are cumulative attacks, also in the morning—and when the convulsions have ceased a gradual prolongation of the interval between the doses; for example, after a few weeks of this treatment the medicine is omitted one day in every six, later for two days, etc., until finally it is given only six days in every two weeks.<sup>1</sup>

Epileptic children must be under constant observation. Opportunity for injury during an attack must be carefully avoided; therefore epileptics should not bathe in public places or be permitted to handle pointed and sharp articles without supervision. I discourage the sending of a child to strangers in the

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<sup>1</sup> I have not been able to convince myself that these intermissions are advisable. A few days of them were sufficient to cause new attacks. That is why I warn against them. My night dose is equal to the sum of the two day doses. All of them are continued through months or years. Frequent combinations are digitalis or strophanthus in heart affections, arsenic to protect the skin and for its general effect.—EDITOR.

country unless there will be the same painstaking care as at home. The choice of an occupation presents great difficulty and usually must result in some easy house employment. A decision in this respect is particularly difficult, especially if the attacks are infrequent, when there is a tendency and talent for some successful trade.<sup>1</sup>

Many paths lead from functional nervous diseases to mental affections. From neurasthenia the road leads over the province of psychopathic inferiority to degenerative psychoses; from hysteria and from epilepsy to the mental diseases which arise from the same foundation.

Mental disturbances are comparatively rare in infancy, and are not much more common after that period. There is, however, an extraordinary limitation of the psychic development which we designate, according to its degree, as imbecility or idiocy. Both of these conditions, as well as all other mental diseases of infancy, will be considered in a special volume.

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<sup>1</sup>See also volume on "Diseases of the Nervous System," p. 1012.

## CONVULSIONS IN CHILDREN

By K. HOCHSINGER, VIENNA

*Muscular spasms of clonic and tonic nature* occur more commonly in infancy than at other periods of life. When dependent upon organic changes in the central nervous system they show no deviation from those of later years except in their greater frequency. Acute and chronic affections of the brain and its membranes, and the toxoinfectious diseases of the central nervous system, for example, tetanus and hydrophobia (Lyssa), produce convulsive seizures at any period of life. In contrast to these *symptomatic* convulsions which occur at all ages there arises in infancy—and particularly at a quite definite period—a second group of hyperkinesias which play a much more dominant rôle, being characterized by *tonic or clonic convulsions* of the voluntary muscles, independent of organic disease of the central nervous system. The forms of spasm just mentioned are inappropriately and indiscriminately designated as *essential* or *idiopathic convulsions* of children and are in contrast with the “*symptomatic*” convulsive conditions.

If the epithet “essential” or “idiopathic” were meant to express the independence of the convulsions in question from other conditions its use would be ill-advised.

The more we learn of the nature and causes of infantile convulsions the clearer does it become that even those convulsive conditions of children which are independent of material changes in the nervous system are no more than symptomatic expressions of other affections which damage the infantile organism. Their spasmogenic action has as yet not been defined in all of its details, but it has been determined with absolute certainty that they are able to generate convulsions in children without previously producing recognizable anatomical lesions in the central nervous system.

To the so-called idiopathic convulsions of infancy belong *eclampsia*, *spasm of the glottis*, and the *convulsions of tetany*, since in a great majority of cases these are apparently substantive affections. But eclamptic attacks without anatomical cerebral lesions also occur in the course and as the direct consequence of other manifest infantile diseases, in which, therefore, there can be no question of an idiopathic character of the hyperkinesia. For example, if a child is attacked with convulsions during and in consequence of an eruption of measles they are certainly not idiopathic but are due to the action of the poison of morbilli upon the central nervous system. In the ordinary use of the term, however, they are not “symptomatic” convulsions, for, so far as is



known, they are by no means dependent upon material changes in the central nervous system.

Accordingly, the division of infantile convulsions into "symptomatic" and "idiopathic" is neither entirely correct nor adequate. In the broadest meaning of the term all convulsions of children are symptomatic, and they must be either dependent upon organic lesions of the central nervous system, therefore "organic," or exempt from such lesions, therefore "functional."

Nevertheless, it must be admitted that several of the functional convulsions of infancy naturally crystallize into other groups in their etiology and in their clinical picture.

In particular there is a great group of convulsions which reveal the attributes of a substantive disease by the typical recurrence of single attacks for a prolonged period and by the *overirritability of the nervous sphere* during the interval free from attacks. These are mostly the cases which have been designated *idiopathic eclampsia of children*, but, according to the present state of our knowledge, will here be termed the *overirritability convulsions of children*.

In a second group of convulsions the individual paroxysm has merely the importance of an *accidental* occurrence. It is an accompanying symptom of some clearly-defined disturbance of health, generally of an acute nature; but the typical repetition of the individual paroxysm, peculiar to the convulsions of the first group, is absent. The attack is either due to a *reflex irritation of the spasm center* (*sympathetic or reflex eclampsia*, Soltmann) or to some deleterious agent acting upon the circulating blood or other fluids of the body (*eclampsia haematogenes*, Soltmann). The convulsions of the last named group occur only while the manifest disease is present. In those of hyperirritability the recurrence of the same form of convulsion is the factor which lends to the seizures the appearance of a substantive affection, and to which they owe the designation "idiopathic." In the hematogenous and reflex eclampsias, however, we are concerned merely with *occasional convulsive attacks* at the onset or in the course of an acute disturbance, as at the outbreak of fever, after injury, from the action of some foreign body, and the like. Here belong also the tonic convulsions of the muscles of the extremities in very ill children in the first weeks of life (*myotonia of the new-born*), in which we are dealing with permanent tonic spasms, dependent for the most part, according to Soltmann's nomenclature, upon hematogenous causes.

In a clinical respect an isolated position is assumed by a small group of infantile spasmodic conditions, namely, *true tetany*, in which tonic intermittent convulsions of the muscles of the extremities of quite definite form, with retained consciousness, are noted. Recent authors maintain that this form of convulsion is dependent upon *disturbances of internal secretion* within the *parathyroid glandular bodies*, the so-called *epithelial bodies*.

In this article we shall concern ourselves principally with *functional convulsions* of children, since these occupy a special position in the nosology of infancy, and in the main present but little resemblance to the pathology of later periods of life. Only in the *first two years* do functional convulsions

occur with great frequency; with advancing years the tendency to spastic attacks of a functional nature constantly decreases. The convulsions due to organic lesions will be described in the course of this article only to such an extent as is necessary to comprehend the nature of convulsions of infancy. They will be briefly dwelt upon in the differential diagnosis.

## I. GENERAL ETIOLOGY AND PATHOGENESIS

In any pathologic irritation which is manifested by the appearance of convulsive conditions the nerve centers may be affected as follows:

1. Through a physiologic increase of stimulation conducted from the periphery to the nerve cells.

2. Through a biologic alteration of the nerve substance, in the broadest sense of the term, in consequence of nutritive disturbances arising from physical, chemical, or toxoinfectious influences.

In an anatomical respect it must further be remembered that convulsions may be produced in three ways, and we must differentiate, according to the region from which the irritation arises:

1. Convulsions due to *direct* stimulation of the *bulbomedullary* centers;
2. convulsions due to *direct* irritation of cortical or infracortical *cerebral* centers;
3. convulsions due to stimulation of sensory centripetal tracts and *reflex* stimulation of bulbomedullary or cerebral ganglion groups.

There can be no doubt that these forms of convulsions which have been demonstrated experimentally and have long been recognized clinically in the pathology of more advanced age, also occur in childhood, for the central nervous apparatus of children, apart from the incompleteness of the medullary sheaths of the conduction tracts, is organized in the same manner as the nervous system of advanced age.

Starting with these premises, the question arises, how may we explain the occurrence of *functional infantile convulsions*. There can be no doubt that in the functional spasms of infancy only those centers and tracts can be concerned in which, under other circumstances also—i. e., experimental irritation or organic disease of the nervous system—the spasmodic irritation originates and runs its course. However, as the convulsive phenomenon in functional spasms does not develop from material alteration of the previously mentioned centers and tracts, as is the case in those due to organic lesions, and a spasmodic irritation is inconceivable with normal function of the parts of the central nervous system in question, there must be some additional factor in functional convulsions of infancy which is equivalent to the spasmodic irritation of an organic lesion of the central nervous system or to one experimentally produced. This agent is to be found in a damage to the central nervous system from various external and internal influences which are conducted to the nervous system during the course of many infantile diseases.

Unless we wish to enter into a discussion regarding the ultimate cause

of infantile functional convulsions it is impossible to proceed without the introduction of an auxiliary conception. This auxiliary conception is the *tendency to spasm*; which cannot be sharply defined, of the first years of life, a "*spasmophilia*," by which the above-mentioned deleterious agents of internal and external nature produce a spastic stimulation in the nervous system in early childhood more readily than at later periods. It must also be added that during the first three years, which form the principal domain of infantile convulsions, this spasmophilia is not uniform and that some forms of convulsion occur only in the first months of life, while others show a preference for the second and third years.

A support for the conception of infantile spasmophilia may possibly be found in the anatomy and physiology of the central nervous system in early infancy. In an anatomical respect we must remember that the central nervous system is not fully developed in the new-born and especially that the *covering of the medullary sheaths of the conduction tracts* and nerve fibers is still *defective* in the first months.

In a functional respect, according to Flechsig, the new-born child, with its almost wholly immature cerebrum, resembles an animal with its brain removed in experiment. Although a great part of the functions, and in particular those that are necessary to life, are maintained without the coöperation of the cerebrum, and although, in the brainless dog, the satisfaction of the lesser corporeal instincts takes place without hindrance, just as in the new-born, nevertheless, for the performance of associated actions and movements the cerebrum, and particularly the cortex of the brain, is indispensable. It may be assumed that the nerve medulla is in intimate relation with the fullest functional activity of the nerve fibres, and that the peculiar sluggishness and helplessness in the movements of the nursling are largely due to the absence of a covering for the medullary sheaths of the axis cylinders. The characteristic tendency of the new-born and of young infants to persistent tonic spasms also depends in great part, as will be explained later, upon the conditions which have already been indicated. Furthermore, we know that the cortical control of the motile apparatus is still incomplete in earliest infancy, and that the central inhibitive apparatus especially—one of the most complicated mechanisms of the nervous system—is yet quite immature.

A third essential factor which has to do with the central nervous system in early childhood is the *rapid growth of the brain*, which in the second quarter of the first year progresses with particular intensity. As in all rapidly growing tissues, a special vulnerability of the brain during this stormy period of development must be assumed.

Therefore it is readily perceptible that if, during this period of rapid growth of the brain coincident with the psychical development, an intense injury of a general nature affects the entire organism of the child, the singularly sensitive nerve substance which is still undergoing development will be particularly implicated.

Thus the ready excitability of the nervous sphere of the child is easily



understood. It is also self-evident that the abnormal irritability of the cerebrum is not so great during the first months of life as later; on the contrary, it only becomes obvious in the second quarter of the first year (Soltmann), since it goes hand in hand with the psychic development which in turn is intimately associated with the anatomical growth of the brain. However, as this *psychical hypersensitiveness*, due to embryonic causes, is still unopposed in the later period of infancy by a strong *inhibitive property* of the brain, it is comprehensible why abnormal stimulation of the nerve centers is so frequently associated with muscular spasms, particularly in the period of life under consideration.

Only by taking into account the anatomical and physiological peculiarities of the infantile central nervous system is the feeble faculty of resistance to external and internal influences explained, which accounts for the appearance, even upon slight stimulation and with insignificant anatomical lesions in the course of the central nervous apparatus, of convulsive conditions which would never become noticeable in older individuals under similar circumstances. *Reflex irritability*, which also undoubtedly exists during early infancy, finds its explanation in the *absence or deficient development of the inhibitive centers* of the cerebrum, which is equivalent to an abnormally great vulnerability of the nervous substance.

Nevertheless there can be scarcely a doubt that in the occurrence of infantile convulsions, in addition to other factors which will be considered in a special chapter of this article, *individual predisposition* plays an important rôle. *Hereditary* influences are especially noticeable. Those of long experience and actively engaged for years in consultation and family practice cannot evade the fact that the children of nervous parents in particular attract attention even in earliest life by an abnormal condition which shows itself in frequent convulsive attacks from relatively slight causes. But here other individual factors also come into question. It must be remembered that convulsions do not appear in all children under similar external conditions. The factors which give rise to convulsions do not act uniformly in all children nor in children of the same age. There are individuals who throughout infancy and until the period of pubescence are seized with convulsions from the most insignificant causes, for example, slight digestive disturbances, mild fever, or even purely psychical irritation, which have absolutely nothing in common with epilepsy, but it is not possible in every case to state exactly why this spasmophilia which is so obvious is a property peculiar only to these children. Although it occurs more commonly in rachitic and artificially-nourished children and those with a hereditary predisposition, nevertheless it may be present in those free of rickets, in breast-fed children, and in those without obvious heredity; therefore, in the theory of infantile spasmophilia there are still many unsolved problems.

In former times all functional hyperkinesias of children were declared to be reflex, but the more extensively the nature of infantile spasmophilia was studied the more obvious did it become that the old theory of reflex convulsions

was unsatisfactory. Also external mechanical irritation involving the sensory nerves (great and sudden changes in temperature, foreign bodies, burns, suppuration, fissures) does not alone suffice for the production of convulsive attacks; on the contrary, such factors are calculated to predispose to their development only when a positive general spasmophilic constitution is present, which is indicated by an increased irritability of the central nervous system, and which is the actual pathologic condition of the case in question. The anatomical and physiological peculiarities of the central nervous system in first childhood may explain the tendency to convulsions, but not the "wherefore" of the spasm, accordingly, not the pathology of the convulsive attack, not the agent which in the individual case causes the transposition of the increased irritability into the convulsion.

If the anatomical, physiological, and embryological peculiarities of the infantile cerebrum, in combination with definite, spasmophilic, external effects, were the only causes of functional infantile convulsions it would then be impossible to understand why all children who are subject to the same external causes are not attacked by convulsions. Although this is not the case, nevertheless, the nature of functional infantile convulsions in its final phases is not yet entirely clear. The conditions to some extent resemble those of *epilepsy*. In this disease also a special pathologic composition of the central nervous system, particularly of the cerebrum—a sort of hyperirritability of the latter—plays the main part, the spasmodic attacks themselves being simply the visible signs of the functional anomaly of the brain, the nature of which has not yet been explained. Experimentally in the animal it has been proven by Brown-Séquard, Westphal and Unverricht that certain traumatic manipulations (severing of nerves, continuous percussion upon the occipital region, electric stimulation of the cortex of the brain) produce a peculiar labile condition of the brain, impossible to define more accurately, which is revealed by frequent convulsive attacks and therefore may be regarded as a sort of artificially-produced spasmophilia. In this respect particularly there is an analogy between epilepsy and the functional convulsions of children.

What these authors have proven in their experiments upon animals is that the labile condition of the nervous system which tends to convulsions is natural in infancy, that is, there is an increased vulnerability of the nervous apparatus which probably in great part owes its ultimate origin to special physiological and anatomical relations.

During the first two years of life this spasmophilia is especially conspicuous. Many authors maintain, however, that in the first weeks or months the peculiar tendency to convulsions does not exist. I cannot coincide with this view but must remark that the spasmophilia of the first weeks of life in general assumes a clinically different form from that of the following period. In the latter months of the nursing stage and during the second year of life there is a tendency to *clonic and tonic convulsive conditions of intermittent character*, while the first extrauterine period is distinguished by a tendency to *permanent tonic convulsions*. Accordingly, functional clonic convulsions of the external muscles

and spasm of the glottis are rare occurrences in the first three months of extra-uterine life. With advancing age eclampsia and attacks of laryngospasm increase and in the second year of life convulsions become more common, attaining their greatest frequency at the end of the first and during the second year.

A reason, perhaps, that eclamptic attacks and laryngospasm are rare occurrences in the new-born and in very young children is the fact that the most important etiologic factors producing these conditions, with which we shall become familiar later, namely, *respiratory noxæ*, in consequence of prolonged residence in impure air, have not had an opportunity of acting upon the nervous system sufficiently long to produce a special hypersensitiveness of the central nervous apparatus. Inversely, permanent tonic convulsions of the muscles of the extremities in babies, in their external representation, are by no means such conspicuous occurrences as are the seizures in later months, and accordingly are often overlooked or at least are appreciated much too little.

With the beginning of the third year of life the tendency to spasmophilia decreases more and more and children who formerly were tormented by convulsions are frequently quite normal after the third year. A small number, however, retain a certain tendency to spasmophilia up to the age of puberty. These are children who, even in late childhood, are invariably seized with convulsions upon the appearance of some acute febrile disease, as a mild tonsillitis or a simple attack of gastric indigestion, reacting by clonic spasms and disturbance of consciousness. Such children very often remain permanently in a state of psychic hyperirritability, and it is an experience gained from long years of family practice—*sit venia verbo*—that the convulsive infants frequently become the nervous and hysterical children of later years.

The predilection for the second, third, and fourth years of life of the more substantive functional convulsions, which according to our view depends upon a permanent pathological hyperirritability of the central nervous apparatus, requires a somewhat fuller discussion.

This fact receives a satisfactory explanation if we remember that the tendency of children to convulsions depends upon an alternate relation between cerebral development, on the one hand, and damaging influences during this period, on the other.

That the convulsive attacks due to psychical hyperirritability do not occur in the first weeks is readily understood, since at this period the brain does not exercise essential psychical function, and also because those damaging influences which in later months give rise to eclampsia and laryngospasm have not had sufficient time to exert their effect.

Between the second and fourth years the nervous system is in an unfavorable constellation. The enormous psychical and cerebral development at this time is coincident with the appearance of one of the most widely distributed constitutional diseases of infancy, *rachitis*, to say nothing of the many influences of infectious, alimentary, and respiratory nature which at this age have had a sufficient opportunity to exert their deleterious effect upon the infantile nervous system.



At the end of the second year, on the one hand, the development of the central nervous system is more complete, and on the other, the deleterious factors of general nature, which have just been mentioned, become more secondary, so that from the beginning of the third year the tendency to convulsions begins to lessen.

From our present views it is sufficiently evident that a uniform basis for convulsive conditions in the first years of life may easily be attained if we constantly bear in mind that infantile spasmophilia depends essentially upon the reciprocal action of the anatomical and physiological development of the brain on the one side, and the influence of certain deleterious irritating factors upon the other. By the same line of reasoning, as we shall see later, the fact may be explained that every period of infancy possesses, I might say, its individual form of convulsion. The first two or three months are characterized by a preference for *permanent spasm of the muscles of the extremities* and the absence of mechanical and galvanic hyperirritability of the nervous system, while the second half of the first year is dominated by *psychical hypersensitiveness*. Accordingly, during this latter period all of those intermittent forms of convulsion are to be found which are associated with clinically manifest *hypersensitiveness of the nervous system*. We shall not go astray if we regard hypersensitiveness of the nervous system as the basis for all of these forms, and certain occasional causes which cannot always be accurately defined as the *causa movens* of the isolated convulsive attack.

In the second and third years a third, clonic and persistent, form of convulsion is added which is likewise associated with hypersensitiveness of the nervous system; the *nodding spasm and spasm of the ocular muscles of children* (so-called *waddling of the head and eyes*).

Functional convulsions in children may be considered from various standpoints.

First, as to the nature of the convulsions. They occur as *clonic spasms* or *twitchings* and as *tonic spasms* or *contractures*.

The contractures, in turn, may be either *intermittent* or for a long time *persistent*. Accordingly we differentiate between *tonic intermittent* and *tonic persistent* or *permanent spasms*. Tonic and clonic convulsions are very often combined.

Their differentiation as to *localization*, that is, according to the muscle regions affected, is very important. Convulsions of the *external muscles*, predominantly clonic in character, are included under the collective term "eclampsia." This variety of functional spasm is in contrast with another form in which the *muscles of respiration*, therefore the internal muscles, are affected, the nature of the convulsion being *tonic*. These are the *respiratory spasms* of children, their principal representative being *spasm of the glottis*.

These two varieties of convulsion, eclampsia and respiratory spasms, are frequently combined in an attack, so that in a clinical respect their complete separation is not always possible.

It must be remarked that in *tonic contractures of the muscles of the extrem-*

ities the intermittent forms are sometimes associated with both of the aforesaid varieties. Only the tonic persistent convulsions of the extremities of quite young infants have no direct relation to the clonic convulsions of children.

Eclamptic conditions, as we shall find later, also occur in very young infants, and it can happen that a child of this age, affected by permanent spasms, may be attacked by eclampsia as well. In such cases, in addition to the permanent spinal stimulation, a cortical irritative condition, either organic or trophic, has occurred, from which convulsions have arisen.

While the respiratory spasms and the tonic intermittent contractures of the muscles of the extremities occupy, under all circumstances, a somewhat isolated position, and convey the impression of a substantive affection of infancy, the clonic convulsions of the external muscles, to which, when they occur in cumulative attacks, the general term "eclampsia" has been applied, are occasional accompanying phenomena of other more manifest acute or subacute diseases (*eclampsia sympathica* and *eclampsia haematogenes* of Soltmann), although in a number of cases they are of a more essential character.

If, however, we enter into a minute investigation of the attacks of eclampsia in individual cases, a differentiation may perhaps be made between the forms of more substantive character and those which are only accompanying phenomena of some other clearly defined disease. The paroxysmal clonic convulsions which occur in the *course of acute diseases of infancy*, and especially in the infections, are almost always confined to the *external* musculature, the *respiratory muscles being very rarely involved*. On the other hand, the eclamptic attacks which occur outside the realm of the diseases of infancy, therefore those of a substantive nature dependent upon general hyperirritability, are almost always associated with spasm of the larynx. Accordingly, I believe that the external clonic muscular convulsions subordinate to other manifest toxoinfectious diseases should be designated "*simple convulsions*," and discriminated from actual "*eclampsia*," which is a combination of clonic external spasms and tonic respiratory spasms and occurs for weeks or for years in irregular attacks independently of other acute diseases.

One fact must be emphasized: Children who suffer from the last-mentioned form, i. e., true eclampsia, or *eclampsia* correctly called by that name may occasionally be attacked with these true eclampsias in combination with respiratory spasms during the course of other diseases, for it is well known that convulsions even of an essential nature preferably arise in infancy during acute intercurrent disturbances of health. It is a conspicuous fact, however, which in my judgment has not been sufficiently appreciated, that the convulsive seizures of infancy which occur secondarily to acute disturbances of health rather possess the character of pure external clonic muscular contractions, while the clonic convulsions of more essential, substantive nature almost never occur without involvement of the internal muscles, especially those of respiration.

Of great importance is the *psychical state* of children during the convulsive attacks. During the simple convulsion as well as in true eclampsia there is a

disturbance of consciousness. Occasionally respiratory spasms, running their course without involvement of the external muscles, are associated with transitory unconsciousness. It is therefore obvious that in the varieties of convulsion associated with loss of consciousness the irritant which produces the spasm diffusely involves the central organ of the nervous system, especially the cerebral cortex. Inversely, in the tonic persistent convulsions of the muscles of the extremities which appear in the first weeks of life loss of consciousness does not occur. In the tonic intermittent spasms of the muscles of the extremities of the late nursing period there is as a rule a disturbance of consciousness. Only in the genuine carpopedal spasms of tetany<sup>1</sup> is there no derangement of the mental condition.

Accordingly, we shall not go astray by regarding the convulsive eclamptic attacks as of *cortical*, and the tonic persistent convulsions of the muscles of the extremities and the true tetanic spasms, respective of their sphere of irritation, as of subcortical or spinal origin. Perhaps even the anatomical region from which the spasm in functional infantile convulsions originates may be accurately determined in the individual variety of convulsions from the clinical picture. In true eclampsia, which is characterized by contractions in widely distributed muscular regions, associated with *loss of consciousness*, the *cerebral cortex* is positively to be regarded as that sphere from which the convulsions originate. Likewise, there can be no doubt that the cerebral cortex is irritated diffusely in all forms of convulsion in which a disturbance of consciousness appears.

In the simple *respiratory spasms* of children a decision of the question as to the central point of irritation is not so easily solved. There are respiratory spasms of children which recur quite often with retained consciousness. To this category belong the milder forms of spasm of the glottis which are regularly accompanied by crowing inspiration in crying. Superficial investigation would seem to indicate an isolated irritation of the bulbar respiratory center, that is, of those ganglion groups in the bulbous which are concerned with the innervation of the vocal cords.

In a large majority of the cases, however, the eclamptic attack begins with respiratory spasms, and a disturbance of consciousness occurs later in association with general convulsions. Here also the explanation might be that there is first a bulbar irritation of the respiratory center, the convulsions being regarded as suffocative spasms which originate primarily from arrest of respiration. We should then have to consider a simultaneous irritation of Nothnagel's convulsive center in the medulla oblongata.

Finally, to explain the great frequency of simple spasm of the glottis in in-

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<sup>1</sup> My view in regard to this point differs essentially from those generally held at present. Most authors indicate that the attacks of infantile tetany are associated with disturbance of consciousness because the entire subject of eclampsia and of respiratory spasms is summarized by them under the conception of "infantile tetany," to which I cannot agree. I regard the tetany of children as identical with tetany of adults, and explicit statements in this respect will appear later:



fancy it might be assumed that, while it originates subcortically, this irritation arises more readily in young children because at that age inhibitions on the part of higher centers are slight.

But the following exceedingly important factors are against this view, that the functional respiratory spasms of infancy are due to localized subcortical irritation:

1. Respiratory spasms of children are always associated with *psychical irritation* and never appear without external cause in a condition of complete mental rest.

2. In cases associated with eclampsia and unconsciousness the latter condition occurs with such lightning-like rapidity in the initial respiratory convulsion that there can be absolutely no question of its being due to an arrest of respiration.

Everything indicates that the functional respiratory convulsions of children are also of cortical origin and that a certain *psychical hyperirritability* is the foundation of these as well as of all other forms of convulsion of later infancy and of early childhood, although they may be seemingly of a more idiopathic character. Therefore, the respiratory spasm is absent so long as an essential psychical activity has not yet arisen. The ultimate cause, however, is to be sought in a *psychical hyperirritability* which only becomes operative with the functional awakening of the brain.

A special tendency to psychical irritability, on the one hand, and a certain deficiency of inhibition through higher centers, on the other, are undoubtedly the characteristics which are the basis of spasmophilia in early infancy. It must be again remarked, however, that this irritability only becomes operative with increasing development of the brain, therefore in the second three months of life. In the first two or three months all forms of convulsion are absent which are dependent upon psychical hyperirritability.

In some of the *peripheral nerves* the degree of hypersensitiveness in certain forms of convulsion may be determined. Reasoning from previous explanations it is readily understood that a certain hyperirritability which may be demonstrated electrically is much more distributed in early childhood than in all later periods.

While it is relatively rare in persons of advanced age and is actually present only in a single form of convulsion, namely, *true tetany*, this measurable hyperirritability is widely distributed in the first years of life and it is not rare in various forms of convulsion in children of two and three years so far as regards a *permanent psychical hypersensitiveness*.

Following the publication in 1878 of Soltmann's experiments on young animals, the cause of convulsions in children was for a long time sought in the deficient development of the psychomotor cortical centers of the cerebrum. At that time this author considered that the cerebral cortex is less irritable in early infancy than in adults and that on account of its incomplete development it was not able to exert an inhibitive influence upon centers more deeply situated. Therefore spasmodic attacks could be produced from these deeper centers,

even with slight irritation, without receiving inhibition on the part of the cortex. According to the present status of our clinical and physiologic knowledge Soltmann's teaching is no longer sufficient to explain the peculiar spasmophilia of nurslings.

The assumption of a lessened irritability of the cerebral cortex in general would presuppose that inhibitive as well as stimulative impulses arise from the cerebral cortex of the new-born to a much slighter extent than in later life.

In so far as we are concerned with impulses dependent upon psychical influences this is correct. But it would be a mistake to conclude that the cerebral cortex of the new-born cannot be stimulated at all. Pathological anatomy teaches that new-born children with capillary hemorrhages of the cerebral cortex in consequence of trauma at birth are very often subject to tonic-clonic cortical spasms. Even unilateral traumatic and hemorrhagic affections of the cerebral cortex produce contralateral convulsive conditions which are no less severe in the first weeks of life than at any other age. From this it is obvious that the cerebral cortex may be directly stimulated in the new-born, and stimulated to the extent that irritations give rise to contralateral spasms of the extremities, notwithstanding the peculiar and even grossly retarded development of the central nervous system at birth. In the new-born child, as is well known, the white cerebral substance is quite deficient in medullary fibers. Only with the fifth extrauterine month is the medullary sheath-covering of the nerve fibers completed. The negative results obtained by Soltmann in experiments upon new-born dogs and rabbits, in which contractions of the contralateral members were produced by electric stimulation of the cerebral cortex, discovered by Fritsch and Hitzig, appear to favor the view that the functions of the cortex do not take place so long as the medullary sheath-covering of the axis cylinders is incomplete. As, however, Soltmann observed that this retarded condition of the cerebral cortex in new-born animals continues until the tenth or twelfth day of life, and from similar anatomical investigations of the development of the medullary sheath it appeared that this immaturity of the central nervous system in young animals about corresponds to the second or third month in the new-born child, he believed himself justified in assuming that in the latter, in the first two or three months of life, there were defects in function of the cerebral cortex similar to those in new-born animals in the first two weeks.

Thus, the view that the property of conduction of the nerve fibers arises coincidentally with medullary sheath production appeared to have much in its favor. Nevertheless, the experimental investigations of Paneth soon became known, which found that in new-born animals, notwithstanding the entire absence of fibers containing medullary sheaths in the motor zone, the cerebral cortex could be stimulated experimentally in the sense of Fritsch and Hitzig. Naturally this altogether coincides with the clinico-anatomical facts previously stated, which show that cortical diseases of the new-born may lead to convulsions in the extremities of the opposite side.

In opposition to these experimental and clinico-anatomical facts Moussous's

explanatory attempts must appear feeble. Adhering to the insusceptibility of the cerebral cortex of the new-born to irritation, this author attributes a special significance to convulsions following traumatic obstetrical hemorrhages of the cerebral cortex, which would indicate that changes of this nature are always associated with rupture of vessels in the soft membranes of the brain, so that the extravasated fluid blood reaches the subarachnoid spaces and flows thither to the most dependent parts. In this way the extravasation comes in contact with the cerebral peduncles and the bulbous, from which regions spasmodic phenomena are said to originate by means of Nothnagel's spasm center in the bulbous.

To enter at this point upon a commentary of such a forced argument is unnecessary. The fact stated by Moussous that a great number of cortical affections, acquired intrauterine, run a latent course in the first weeks of life and do not give rise to recognizable symptoms until later is by no means opposed to the possibility of a spasmodic irritability of the cerebral cortex in earliest childhood, as at this period of life many cases of encephalitis run their course with convulsive paroxysms.

Therefore, if the view that the cerebral cortex of the new-born cannot be stimulated is proven to be erroneous, it is quite as positive that in early infancy the *inhibitive and regulating* action of the cerebral cortex is incomplete. The peculiar conditions of the muscle tonus of new-born children, which later will be the subject of a comprehensive discussion, constitute the most valuable confirmation of this law, which is further strengthened by the animal experiment.

From the foregoing the very important clinical conclusion may be drawn that in clonic convulsions of the new-born and of young nurslings we must be exceedingly cautious in assuming a functional nature. In these early stages of life, just mentioned, shaking spasms much rather indicate an organic cerebral disease than a purely functional irritation of the central nervous system.

In regard to the reflex nervous apparatus of early childhood it may be stated definitely that by no means is there the degree of completeness that attaches to later life, and that especially the connection between the cortical and infracortical centers do not as yet reach that development in a functional respect which is present in the mature individual. Inhibition and association are inadequately developed in the first years of life. The confirmation of the reports in literature of numerous anencephalic new-born infants whose movements awaken a coördinated impression cannot alter this fact.

In these infants there were in particular normal respiratory movements, and the acts of deglutition and suckling, of crying and resistance occurred promptly, which in any event demonstrates a certain independence of definite reflex acts in early infancy from cortical innervation. It is true, this merely proves that individual movements necessary for the daily processes are regulated without the action of higher centers, while complicated acts are coördinated, inhibited and associated only with the increasing development of the brain. Sufficient evidence of this is the celebrated experiment of Goltz, who kept a dog alive for eighteen months after the brain had been removed, and



found that the animal could satisfy without difficulty its bodily requirements which were necessary for the maintenance of life.

The nature of functional infantile convulsions cannot be understood correctly without entering into the *physiology of the muscular movements*.

We must be quite clear as to what is understood by muscular contraction and muscular relaxation and as to the varied muscle action in infantile convulsions, the clonic and the tonic muscular contractions, and the prolonged spasms. We must also be in position to decide the question: How is the action of *stimulating* and of *inhibiting* nerve fibers expressed upon their terminal organ, the *musculature*.

Of great value in this explanation, which at first sight appears to be difficult, is the theory of Kassowitz, given in the third volume of his *Biology Relating to the Nature of Muscle Movement*, which, like his entire biologic conception, is founded on the principle that *all activity depends upon the formation and destruction of the protoplasm*.

According to Kassowitz the so-called *relaxation* of the muscle, for which a more apt term would be its *elongation*, is just as *active* a process as the *contraction* or *shortening*. Both of these functions depend upon the activity of separate nerve fibers which, in the voluntary and transversely striated muscles, as a rule, pass conjointly to the same nerve trunk.

The muscle fibers, as is well known, consist of two protoplasmic substances, differently constructed—the actual *fibrillary substance*, which Kassowitz designates *myoplasma*, and the surrounding intermediary substance, the so-called *sarcoplasma*. By the well-founded view of Kassowitz that each of these substances is separately innervated, the entire problem of muscle movement and of inhibited movement is explained in a simple manner. With each nerve stimulation conducted to the fibrillary substance destruction occurs within the myoplasma, which must result in a shortening of the muscle in the axis of its fibrilli, while as much sarcoplasma is always constructed during the contraction as equals the amount of destroyed myoplasma.

This causes the muscle to become thicker and shorter.

Again, sarcoplasmic substance is destroyed by the elongating irritations of innervation. This causes narrowing of the muscle, and because the products of disintegration of the sarcoplasma serve to construct the myoplasma, the fibrilli gain just as much in extent in the longitudinal direction as the sarcoplasma has regressed in the vertical direction.

This would explain why the volume of the contracted muscle does not become less than that of the relaxed or elongated muscle.

Therefore, only by Kassowitz's interpretation is it obvious that, instead of becoming harder and firmer, the muscle is made shorter and thicker by the processes which take place in the fibrillary substance as the result of irritation. It only becomes more tense during contraction when its shortening is hindered by fixation of its ends, as is always the case in the uninjured living muscle. In the extirpated muscle the consistence in artificially produced contraction and elongation is always the same.

The antagonism between destruction and construction of both of the muscle substances is explained by Kassowitz, in that the destroyed product of disintegration and the water of expansion are always utilized in the development of the other substance. Thus, the destruction of the fibrillary substance leads to longitudinal contraction of the muscle, but to thickening in its vertical axis. The reverse occurs in elongation. The change of shape in the elongating phase of the muscular movement depends upon the destruction of the sarcoplasma and upon the restoration of the fibrillary substance in the phase of shortening of the muscle.

The problem of *inhibition* is in intimate relation to *elongation of the muscle*.

According to Kassowitz the inhibition of the contraction of voluntary muscles does not depend upon an incomprehensible destruction of the contraction stimulation conducted to them but upon an isolated innervation of the sarcoplasma by which is brought about a destruction of this contractile substance, a transposition of the liberated water of expansion to the assimilable products of destruction in the fibrilli, a reconstruction of the fibrillary substance and with it an elongation of the muscle fibers.

Charles Bell and C. Ludwig have already assumed and studied this double innervation of the muscular fibers and a direct action of *inhibitive nerves* upon the muscles, and have concluded that the destruction of muscular substance occurs through the excitomotor, the assimilation through the inhibitive nerve tracts.

This conception, however, is shattered by the circumstance that the processes of destruction and construction which lead to a change of shape of the muscle are referred to one and the same substance, so that the various phases of the muscular contraction would not be rightly interpreted.

Only by Kassowitz's theory of the existence of *separate innervation* for each individual muscle fiber, whereby those in connection with the myoplasma produce shortening or contraction, and those in association with the sarcoplasma bring about elongation or relaxation, can the effect of inhibitive fibers upon the muscle substance be understood.

Accordingly, the impulse which leads to strong muscular contraction depends upon a single innervation of the fibrillary substance, and this can only occur through a voluntary central innervation. Every artificial stimulation of the muscle nerves would have to stimulate both varieties of fibers, as the irritating and inhibitive fibers proceed in the same nerve trunk. Therefore voluntary muscular movement is always more intense than any artificially-produced contraction, because in the latter both muscle substances are brought into play and there is always a weakened retraction on account of the simultaneous stimulation of the elongating fibers. That muscular contraction occurs at all with artificial (electric) stimulation is due to the *greater sensitiveness of the fibrillary substance of the muscle than of the sarcoplasma*.

Therefore, stimulation, either direct or transmitted from the muscle nerve of both muscle substances, will cause first a reaction of the irritable myoplasma

which will result in contraction. This muscular shortening will be relieved by elongation when the irritative destruction in the sarcoplasma, which occurs later, has reached a sufficient degree. If, however, stimulations play upon the muscle in rapid sequence the latter effect alone will be observed, as the elongation which follows the irritative destruction of the sarcoplasma always occurs later than the contraction due to the destruction of the fibrillary substance.

The destruction in the sarcoplasma which succeeds the disintegration in the myoplasma will not be especially prominent as regards its elongating action if the stimulations acting upon the muscle follow one another in too rapid sequence. Then the muscle-shortening effect of the permanent stimulation will reach its total and conceal the elongating action of the destruction of the sarcoplasma.

Upon this the nature of the *permanent contractions* depends. But it must also be remarked that the degree of shortening produced by voluntary contraction never occurs by a permanent shortening due to a direct irritation of the muscle or nerve, because irritation acting upon the sarcoplasma, which causes the muscle elongation is never entirely excluded under these circumstances and is to a greater or less extent opposed to the shortening effect of the destruction of the myoplasma.

In passing to the pathology of functional infantile convulsions much that was previously puzzling will be explained in the manner of appearance of these neuroses of infancy.

There can be no doubt that in early infancy the influence of inhibitive nerve centers in the reflex movements is much less than in later life. In all stimulation originating from the center the irritation which leads to destruction of the myoplasma will prevail over that which causes destruction of the sarcoplasma and which is associated with elongation of the muscle. If, therefore, in a general irritation of the nervous system, the stimulating and inhibitive centers suffer to an equal degree, the effect of the former upon the musculature in early childhood will always preponderate. This is the reason of the hardness of the muscles in convulsive attacks and the very much more prominent contractions than relaxations in infantile spasm. As, however, in a general stimulation of the cerebral cortex, irritations are conducted to the myoplasma as well as to the sarcoplasma of the muscles, as already stated, we note in all of those forms of convulsion in children which are associated with disturbance of consciousness, therefore in which the entire cerebral cortex is stimulated, the occurrence of *muscular spasms* which are the equivalent of an alternate shortening and elongation in the muscular regions involved. On the other hand, those convulsive attacks of infancy with retained consciousness, in which the irritative center is subcortical or spinal, possess a predominantly tonic character. Then we are concerned with permanent contractures because only those fibers are notably involved which conduct the irritative destruction to the myoplasma.

This latter condition is especially prominent in the permanent contractures of the extremities of the new-born. As the reflex acts of the first weeks of life



are almost invariably of a spinal nature and the nervous inhibitive influences are not yet operative, we find almost exclusively, in this period, tonic spasmodic conditions.

The conditions are similar in the true tetanic convulsions of infancy, the origin of which must likewise be considered without an implication of the motor centers of the brain.

In eclampsia, on the other hand, there is a general irritation affecting the cerebral cortex, as is shown by the profound disturbance in consciousness, in which excitomotor as well as inhibitive ganglion groups are stimulated. Here, accordingly, the fibrillary substance as well as the sarcoplasm are affected by irritative destruction and the variation in their clonic character explains the appearance of the convulsions.

Of special value are the experimental findings of Soltmann in the peripheral nervous system of new-born animals. This author, who experimented on rabbits, cats, and dogs, found that the *muscle diagram* of new-born animals, which could be obtained by electric stimulation of the nervous and muscular apparatus differs essentially from that of older animals. In new-born and quite young animals the muscle diagram showed curves of slow ascent in a slanting direction with a right or oblique angle at the top; the apex, therefore, was flat, and the descent of the curve gradual. In other words, the muscles of new-born animals, when under stimulation of the motor nerves, remain for a long time at the maximum of their contraction, and the relaxation occurs by slow degrees. The muscle diagram of the new-born animal is identical with that of an older and *exhausted* animal. To originate a tetanic contraction in the new-born animal a far less frequency of stimulation is necessary than in the adult animal. In the latter the tetanic muscular contractions appear only after seventy interruptions of the current per minute, while in the new-born animal they occur after sixteen interruptions. Soltmann quite correctly thought that he had found the key to these peculiar conditions of muscle irritability in the fact that in the first weeks of life of the child pure clonic convulsions are more rare than tonic spasms.

The investigations of C. and A. Westphal have furnished valuable information regarding the conditions in the new-born child. Quite similarly to Flechsig's reports of the central nervous organ in early infancy these investigators found an immature medullary development of the peripheral nerves in the first stage of life. In the first extrauterine weeks the peripheral nerves consist almost exclusively of nonmedullated nerve fibers; not until the third to the sixth week is a marked medullary covering of the axis cylinders in evidence. Quite parallel to these conditions is the electric contractility. The incomplete structure of the nerve fibers also corresponds to a very sluggish electric irritability which continues until about the sixth week and then gives way to normal values.

The results of the investigations of Soltmann and of both Westphals closely coincide with Kassowitz's teachings regarding muscle movement. That the curve of contraction is sluggish in the new-born, that the galvanic irritability

of the muscle nerves in this period of life is decreased, is unquestionably the consequence of a deficient development of the nerve medulla, which is to be regarded as a reservoir for the metabolism of the nerves during their function.

The exaggerated tonus of the muscles of the new-born child, which we shall consider in detail later, is readily explained by the slight activity of the elongating innervation in the earliest epochs of existence.

If, as Kassowitz assumes, the elongation, i. e., the irritative destruction of the sarcoplasma of the muscles, depends upon the action of inhibitive fibers, it must be clear from previous explanations that in the new-born that condition of the muscle which originates from impulses of innervation conducted to the fibrillary substance must largely preponderate, and thus the physiologic hypertonia of the muscles of the new-born is understood.

From these considerations the prevalence of tonic convulsions in earliest life also becomes comprehensible. Here the elongating innervation of the inhibitive fibers, associated with the destruction of the sarcoplasma, is all the more secondary because normally the stimulation of the fibrillary substance, which produces contraction of the muscle, preponderates.

## II. VARIETIES OF INFANTILE CONVULSIONS

Before proceeding to the clinical consideration of convulsions of infancy we must undertake their serviceable classification.

It has already been remarked that the cases in which the convulsive paroxysm is merely an *occasional* phenomenon of some sharply defined acute or subacute disturbance of health are to be differentiated from those cases in which, at short or long intervals for months or years, convulsions of a special kind recur *continuously* in the form of attacks, while in the interval a *permanent hyperirritability* of the central nervous system is manifest.

As was previously stated, the first-mentioned principal group will be designated "*occasional convulsions*" of children and is to be placed in a certain contrast to the second group of infantile convulsions for which the serviceable term "*hyperirritability convulsions*" will be employed.

In the occasional convulsions of children two subdivisions must be recognized:

1. *Simple convulsions or simple clonic spasms*; and 2. *Myotonia of the new-born and young infants*.<sup>1</sup>

The hyperirritability convulsions include *respiratory spasm and true eclampsia, tetany* and—with some reservations—the *nodding spasms* of children.

### I. OCCASIONAL CONVULSIONS OF CHILDREN

Those forms of convulsions may be briefly designated as *occasional convulsions which occur in association with acute or subacute disturbances of health and in which during the intervals free of attacks no symptoms of hyperirritability*

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<sup>1</sup>See also volume on "Diseases of the Nervous System," p. 934, *et seq.*

bility of the nervous system are present. The convulsive conditions with which we are now concerned, provided we are dealing with children beyond the first three months of life, are predominantly of a *clonic* nature, affect primarily the *external* muscles, are *not* associated with spasm of the glottis, and are *transitory*—disappearing after the acute disease has run its course. They may be *quite properly designated* as “*simple clonic spasms or convulsions*” and for the present will be considered separately.

#### A. SIMPLE CLONIC SPASMS OR CONVULSIONS

Although it has been stated that the spasmodic attacks under consideration are of a transitory nature and are always associated with definite and clearly demonstrable acute or subacute disturbances of health, it is not to be understood that these convulsions cannot recur repeatedly in the same individual. On the contrary, the attacks may reappear in various acute diseases affecting the same child, although they are then dependent upon the new affection. Examples of these are the *febrile convulsions* of infancy. As is well known, there are children who for many years suffer from an acute spasmodic condition upon the occasion of an outbreak of fever. The entire realm of the so-called sympathetic or reflex eclampsias of the older authors is included here, and also Soltmann's eclampsia haematogenes in so far as it pertains to the *infantile convulsions due to toxic causes* which are soon to be considered in detail.

The convulsive paroxysms under discussion are always associated with *loss of consciousness* but are only exceptionally introduced by prodromes. Especially in the occasional convulsions of children of reflex origin the muscular contractions are very often noted immediately. The attack frequently begins with a lightning-like change in the color of the face and in the physiognomy. The child first becomes pale and wholly apathetic. This premonitory condition lasts but a few seconds and is often accompanied by contraction of the muscles of expression. The actual convulsion then occurs, namely, severe spasmodic muscular contractions of the extremities, always associated with complete *unconsciousness*.

The convulsions of the muscles of the extremities are revealed by severe spasmodic flexion and extension of all the limbs. The entire musculature of the extremities and the muscles of the abdomen and back are more rigid than normal, and the head is usually thrown backward; as a rule the muscles of the face contract spasmodically in the same tempo with the muscles of the extremities.

The respiration is usually increased during the spasm and is sometimes irregular and remittent; the expiration is spasmodic. Occasionally there are tonic-clonic spasms of the diaphragm *but never the peculiar spasms of the larynx* characteristic of the hyperirritability convulsions which will be described later. Corresponding to the profound disturbance in consciousness the cutaneous sensation is lost. The tongue is sometimes shoved convulsively forward and backward but is usually pressed against the hard palate, the mouth is closed spasmodically, the muscles of the jaw and of deglutition are contracted, and clonic convulsions of the masseters are occasionally observed.



The duration of the individual attack varies from several seconds to hours. In prolonged spasm there is always a transitory moderation of its intensity.

*Febrile convulsions* sometimes occur in a brief attack at the beginning of fever; occasionally they recur throughout the course of a febrile affection and are regenerated with each exacerbation of the fever. These initial febrile convulsions of childhood were often compared genetically with the *rigors* which characterize the onset of many febrile infectious diseases of later life.

In some febrile infections the spasmodic attack recurs several times, but is always associated with a definite, sharply-limited phase of the disease. For example, in *measles* the spasmodic attacks are observed at the onset of the prodromal fever and are renewed upon the outbreak of the exanthem. Convulsions sometimes occur in *varicella* upon the appearance of the first efflorescences and with each further outbreak of nodules. In all *septic infections* of nurslings convulsions which attend the exacerbations of the process and are especially associated with *pyoderma* are the rule.

With the disappearance of the spasm-producing disease the attacks which I have designated occasional convulsions disappear unless, prior to the disease which has passed, the child has suffered from spasms which belong to the realm of hyperirritability convulsions. With these convulsions, however, *spasm of the larynx*, as a rule, is associated—a condition which will be described later.

However, we cannot altogether reject a certain relationship between the occasional convulsions of childhood and true eclampsia associated with constant psychic hypersensitiveness, as some of those children who have suffered from hyperirritability convulsions from early infancy are subject to convulsions in later childhood upon the most varied disturbances in health.

The majority of the occasional convulsions of infancy depend upon the action of *toxic products* upon the central nervous system, but it must at once be remarked that this does not refer merely to poisons which have been introduced from without, in the restricted sense of the term, but that also an important rôle must be attached to those poisons formed within the organism, in particular the *bacterial toxins* in the *infectious diseases* and the *autointoxication* occurring in *intestinal diseases* of children.

The infantile brain is much more susceptible to poisons than that of the adult, as may be concluded from the peculiar course of *exogenous poisoning* in infancy. Thus, intoxication with alkaloids, with carbolic acid, with iodoform, with potassium chlorate, etc., and especially alcoholic intoxication in early infancy, very frequently give rise to convulsions, while in the adult such a condition is altogether exceptional. Under these circumstances it cannot appear strange that the intoxications arising within the infantile organism, particularly those which originate from bacterial toxins and those which develop within the intestinal canal, give rise to quite different symptoms than would appear in later periods of life.

First we shall describe the occasional convulsions of childhood which occur in connection with accidental *exogenous poisoning*, since the mechanism and the picture of the convulsion may be regarded as representative of those intoxi-

cations which arise in the interior of the organism from poisons due to the infectious diseases and from gastrointestinal autointoxication.

*Alcoholic intoxication* is of special importance in children, particularly in nurslings, in consequence of the transmission of alcohol in the milk of the wet-nurse. The transmission of alcohol which had been introduced into the milk of the nursing woman through the mouth was demonstrated experimentally by Klingemann, Rosemann and Nieloux. Convulsions due to alcoholic intoxication in nurslings following the ingestion of spirits on the part of the nursing woman have been repeatedly observed (Demme, Combe); after alcoholism in the wet-nurse had been discovered and corrected the symptoms of intoxication in the child and the convulsions always ceased. The practical employment of this experience would be the complete withdrawal of alcohol, even in the form of beer, from the diet of the nursing woman.

In older children convulsions associated with alcoholic intoxication have very frequently been observed after the ingestion of spirits. In my experience the convulsions in acute alcoholic intoxication occur either as the precursors of a *comatose stage* or the child first falls into a deep sleep and suffers from the convulsive paroxysm later, during the soporose state. From my own observation and from what I can gleam from the literature, the laryngeal muscles are never involved in these alcoholic convulsions of childhood.

Practically of great importance is *lead intoxication* in early childhood, since this condition is very frequently associated with convulsions and, as a rule, is due to the *external* employment of *medicaments containing lead*. Especially has intoxication associated with convulsions been noted after the employment of Hebra's *diachylon salve* for extensive eczema of the face and head (Hahn). Putnam has shown, in a very interesting article upon lead intoxication in infancy, that these intoxications are much more frequently associated with convulsions in children than in older individuals.

Quite similar statements are true of *opium poisoning* in children. After the employment of opium in early infancy tetanoid tonic convulsions as well as shaking spasms have been observed. Here, as in acute alcoholic intoxication, the convulsions may precede deep coma or occur during the soporose condition. In cases of fatal termination they may appear in a cumulative form prior to the terminal arrest of respiration. That even minimal doses of opium may be serious to infants is a well known fact, as well as the particular frequency in children of this age of an association of convulsions with opium poisoning.

Noteworthy in practice are the convulsions due to *santonin poisoning*. The *vermifuge wafers* which are given to children and which are readily obtainable from the apothecary, have given rise to convulsions after the ingestion of a dose containing 1 decigram (1.54 grains) (Binz). In a case observed by Demme a dose of 1 decigram of santonin given daily for four days was sufficient to produce poisoning with severe convulsions in a child aged six years.

These drugs do not exhaust the list of chemical agents which produce intoxication convulsions in children. *Belladonna* and *mushroom poisoning*

may be associated with convulsions and among the drug intoxications which are combined with attacks of spasm must be mentioned *carbolic acid* and *iodoform* poisoning in the new-born and intoxication with *strychnin*, *bromoform*, and *male fern*.

Poisoning by *antipyrin* and *phenacetin* may produce convulsions in children, and especially upon the appearance of drug exanthemata.

A case of poisoning from *balsam of Peru* in a child aged six years, who succumbed to convulsions, has been reported by Lohans. The poisoning occurred by suckling at the nipple of the mother, which had been anointed with the balsam.

We will now consider the *endogenous toxic convulsions*.

As paradigmatic of this form of spasm we may consider *uremic* convulsions because they occur at all periods of life, even in earliest childhood. It is quite certain that some of the convulsions observed in severe intestinal affections depend upon a uremic basis, as Hohlfeld was able to demonstrate the constant presence of albuminuria and casts in severe gastroenteritis of infancy. Uremic convulsions are more common in late childhood and especially in that period at which scarlatina is most prevalent.

Among the occasional spasms of endogenous toxic nature of infancy *febrile convulsions* occupy the foremost rank in frequency and importance. In the clinical description of simple convulsions we have already referred to their nature and the manner in which febrile diseases are associated with spasm. It need only be added that there is no febrile affection of infancy in which convulsive attacks do not occasionally occur, and that they have been observed throughout the entire period of childhood.

It is true that febrile convulsions show a preference for the first three years of life, and the rise in temperature, therefore the hyperpyrexia, can by no means be regarded as the only cause of the convulsion, since the paroxysms are frequently seen in affections which show an elevation of temperature only slightly above the norm. For example, initial convulsions are frequent in mild attacks of influenza, especially in infancy, or in light cases of angina tonsillaris with but little rise of temperature. Accordingly, the convulsive irritation cannot be considered otherwise than due to the action of the toxic products produced by the infectious organisms, and here it must be remarked that the virus of certain affections has a special predilection for convulsions. In the pneumonia of children particularly, and in the acute exanthematous diseases, convulsions are a frequent initial symptom.

An important rôle is assigned to *gastrointestinal autointoxication* in the pathogenesis of infantile convulsions. Long before the pioneer work of Bouchard—"Leçons sur les autointoxications dans les maladies" (1887) it was known that acute and chronic gastrointestinal disturbances of infancy might be the cause of convulsions. Indeed, in the etiology of infantile convulsions there is scarcely another fact so indisputable as that intestinal diseases give rise to spasm. In the last few years, however, we have proceeded too far, in that we have not been content to consider the occasional convulsions of children as



dependent upon autointoxication, but have regarded the so-called idiopathic, or, as they have been termed, the hyperirritability convulsions, and the condition of hypersensitiveness due to them, as an autotoxic sequel of gastrointestinal origin. This standpoint was especially maintained in the recent dissertation upon convulsions by D'Espine and Moussous at the French Medical Congress at Toulouse, and some modern German authorities, as Thiemich, Gregor, Finkelstein, Lange, Fischl, incline to the same or a very similar view.

At another point we shall discuss the correctness of this theory.

*Gastrointestinal disturbances* occasionally give rise to convulsive attacks, not only during infancy but also in later childhood. That children of six or seven years, or even beyond, may be attacked by convulsions from overloading of the stomach is well known to every practitioner, as well as the fact that *constipation* and the *irritation of worms*, especially the *wandering ascarides*, may produce the paroxysm. However, in the spasms due to the three last-named conditions, it is doubtful whether merely reflex processes or those of toxic nature are the cause of the convulsion. Of great importance and less uncertain in regard to their genesis are the spasms occurring in the *infectious forms of gastroenteritis* of the nursing period, immaterial whether we are dealing with endogenous or exogenous infection of the intestinal canal. In *acute choleriform gastroenteritis* convulsions occur at the onset of the disease as well as in the algid stage, and even in the later period of inflammatory reaction. Here the appearance of convulsions in various stages of the malady is dependent upon varying circumstances. In the period of acute choleriform diarrhea the dehydration of the tissues and especially of the nerve substance (Marshall Hall's hydrocephaloid) is regarded as the cause. In the later stages the general intoxication is made responsible for the convulsions. In fact for all forms of spasm occurring in the course of gastrointestinal disease in nurslings, the toxic-infectious factor is prominent, no matter whether it be due to the action of bacterial toxins which develop during the fermentative processes in the gastrointestinal canal, or whether functional changes of the liver and kidney and a disturbance of the intermediary metabolism dependent upon this are operative.

The convulsive condition which appears in the course of gastroenteritis may occur in paroxysms in the abrupt form of severe shaking spasms of the muscles of the extremities, or a clinical picture may arise and remain for days which possesses a certain degree of similarity with true basilar meningitis (*meningismus*). It must not be overlooked that in the course of severe gastrointestinal disturbance organic changes may also take place in the substance of the central nervous system and give rise to convulsions, which must be considered in a differentio-diagnostic respect. The appearance of acute meningitis in consequence of gastroenteritis of nurslings, as a part phenomenon of the same general infection, has been demonstrated by Concetti, who, in four cases of this kind, found the *bacterium coli* in the *cerebrospinal fluid*.

Clonic convulsions are more rare in the chronic digestive disturbances of infancy associated with *atrophy* than in the acute forms. Here a *rigidity of the entire musculature of the extremities*, similar to that of tetanus—the *myo-*

*tonic type*, according to my nomenclature—is the more common the younger the child. The enterogenous athrepsia of nurslings is in many cases associated with the anatomical lesions of the kidney and liver, which undoubtedly causes a deficient function of these organs and, further, interferes with the intermediary metabolism. Occasionally, in the acute and subacute forms of gastroenteritis of infancy quite similar conditions come into question. In this way a sort of *toxemia* may arise which, with an acute flooding of the organism with poison, causes convulsive attacks and in chronic intoxication produces a tetanic muscular rigidity, and also explains the characteristic coma with muscular rigidity in these nurslings in the terminal stage of the affection which Czerny and his pupils regard as the result of an endogenous acid intoxication.

As to the part played by *constipation* in the convulsions of infancy, the importance of this pathologic condition as a causal factor has been greatly exaggerated. But there is a condition due to constipation, namely, *fissure of the rectum*, in which actual convulsive attacks associated with loss of consciousness may occur in connection with defecation. Here the great painfulness of the ulceration, which is increased by straining upon the sphincter muscle during the act of defecation, must be regarded as the factor which gives rise to the convulsion. In addition we occasionally see transitory, or I might say, acute high-graded constipation, probably due to autointoxication, which may cause convulsions in older children.

A word in regard to the relation of *helminthiasis*<sup>1</sup> and infantile convulsions. According to Schmidt's statistics, mentioned by Moussous, relating to 1,160 children, 56 per cent. of these children were the hosts for various forms of intestinal worms.

On account of the great frequency of entozoa in the infantile intestinal tract, convulsions should be very much more common in children, if there were an intimate relation between helminthiasis and convulsions, than is actually the case. In my opinion a connection between helminthiasis and convulsions can only be maintained with certainty in regard to *ascarides*, the wandering of which into the various cavities of the body gives rise to nervous reflex spasms likewise as other foreign substances. I have never been convinced of the association of convulsions with *tape-worms*, except that attacks might occur during the expulsion of a worm or after the administration of purgatives to expel the segments of the parasite.

It must be mentioned that the convulsions arising from intestinal worms have been regarded as of toxic origin, in which the poisonous products of the parasitic organism are ascribed a decisive rôle.

We must now turn to a brief discussion of occasional convulsions of infancy due to *reflex* causes.

There can be no doubt of the occurrence of convulsive conditions due to reflex causes in infancy, and a rapid *overloading of the stomach* or the movement of *intestinal parasites* in the bowels may give rise to convulsions in older chil-

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<sup>1</sup> Which is more frequent in Germany where children are often kept on coarse or uncooked food than with us.—EDITOR.

dren, but it is necessary to enjoin caution against assigning a place to *dentition* in the etiology of infantile convulsions. Even in the works of modern authors we read of reflex spasms which are attributed to the rupture of a tooth. It cannot be emphasized too strongly that cutting of a tooth is a normal process of growth, that there is no sudden rupture, as occurs in perforation of an abscess, but that on the contrary, the gum gradually atrophies over the growing tooth without injury, hemorrhage or erosion. To Kassowitz<sup>1</sup> particularly is due the credit of proving the untenableness of the teaching of convulsions of dentition, and explaining with great enthusiasm and skill that convulsions which appear in children during the period of dentition are not due to teething but are dependent upon quite different conditions (rachitis, febrile diseases, and the like) which, upon minute investigation, can always be determined without much difficulty.

Certain reflex accidental convulsions occur in children in connection with *cutaneous irritation*. The convulsions following burns, injuries, contusions, and acute inflammations of the skin belong to this category. The spasms which occur in severe phimosis and retention of urine, as well as in polyps and fissures of the rectum, and those dependent upon incarcerated testicles in the inguinal canal, belong to the realm of reflex accidental convulsions. That toxic influences may also be responsible for convulsions arising in consequence of burns and widely distributed cutaneous inflammation requires no special remark.

Among the *physical* factors which are capable of producing convulsions in children we must consider the effect of *excessive cold* or *heat*, although no definite reports in regard to such an outcome are at hand. It is known, however, that in adults epileptiform convulsions occasionally appear after sunstroke.

The forms of convulsions which occur in the course of *pertussis* and *congenital disease of the heart* occupy a somewhat isolated position. Here it is no longer possible to speak with absolute surety of functional convulsions, as venous stasis is always present, therefore an overfilling of the brain with blood, which may be a sufficient anatomical substratum for convulsions, quite apart from the fact that in both of these affections *capillary hemorrhages into the substance of the brain* are not uncommon.

It is well known that very young children, especially nurslings, are not infrequently attacked by convulsive spasms in the course of *whooping-cough* and that these general clonic convulsions occur in connection with the coughing paroxysm. They appear only in severe cases and then usually at the acme of the disease. If a convulsive attack once appears in the course of pertussis, in all probability it will not be the only paroxysm during the progress of the malady. In infants these attacks are of ominous import and many children

<sup>1</sup> Kassowitz will decline this compliment (one of many) as undeserved. Another Vienna author, Fleischmann, wrote on the same subject and in the same spirit 40 years ago. He again refers to Jacobi who in 1862 published his "Dentition and Its Derangements," a series of lectures first published in *American Medical Times*. In that book some authors are quoted who before Kassowitz, before Fleischmann, and before Jacobi contradicted the popular fallacy of attributing undiagnosed convulsions to dentition (or worms).—EDITOR.



suffering from pertussis succumb when convulsions arise. Moreover it must be remembered that the convulsions occurring during whooping-cough are not merely simple functional disturbances of the central nervous system but in many instances are due to severe intracranial disease, especially *extravasations of blood* into the meninges and into the substance of the brain—a condition which is liable to occur in severe cases (See the recent comprehensive report of Neurath on “Die nervösen Complicationen und Nachkrankheiten des Keuchhustens,” Vienna, 1904.). In older children who suffer from pertussis the suspicion of intracranial hemorrhage is particularly justified when they are attacked with convulsions and loss of consciousness. It is true that children who are affected by a permanent hyperirritability of the central nervous system are readily subject to convulsions during whooping-cough, so that the paroxysm of cough is merely the exciting factor of the eclamptic attack, which in children of this category may arise from other conditions. These attacks, however, are always associated with characteristic respiratory spasms.<sup>1</sup> They are especially to be feared in whooping-cough and are often the cause of sudden death.

Quite similar convulsive paroxysms as in whooping-cough are noted not infrequently in infants with *congenital disease of the heart and cyanosis*, particularly when they suffer from an acute affection of the respiratory passages.

*Intermittent clonic muscular spasms of the new-born* require special consideration. It has been repeatedly stated that the greatest frequency of clonic infantile convulsions, not excluding occasional convulsions, occurs in the *first two years of life*. In the *first few weeks* clonic convulsions are much rarer than at later periods. The opinion at one time prevailed that *convulsions in the new-born* were always the result of trauma of the brain during birth, and was based upon Soltmann's report that simple reflex or hematogenous irritations were not capable of producing muscular contractions in the undeveloped cerebral cortex of the new-born. Recent observations, however, have taught that at this age the most varied internal and external factors may act upon the nervous system and produce muscular spasms.

First, the not uncommon *terminal convulsions of asphyxiated new-born infants* are to be mentioned, which may depend upon venous stasis as well as excess of carbonic acid, from which an irritation of spasmogenic ganglion groups arises. As a matter of fact death not infrequently occurs in the first months of life from convulsions in the most varied diseases. If we examine the statistics of convulsions collected by various authors very great contrasts will be noted in regard to the participation of different ages and particularly of the first weeks. Anyone who investigates the clinical material of hospitals for the manner of death of the new-born will find that convulsion is often the terminal

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<sup>1</sup> And consequent disorders of circulation in the brain and elsewhere. Sudden death is not so frequent as the very many cases of paralysis, hemiplegia, epilepsy, idiocy, also mental backwardness attributable to whooping cough. Severe attacks should be stopped, by chloroform if necessary; for each may prove fatal. Such instances prove the criminality of the indolence of allowing whooping cough to keep on for months—it being “a self-limiting disease”—in place of relieving it in as many weeks.—EDITOR.

act, while physicians who attend young children after the first weeks, when they are affected by various diseases, will state that clonic convulsions among these patients are not of common occurrence. All severe infections of the new-born, the septic diseases of the mouth, umbilicus, intestines and lungs, Winckel's and Buhl's diseases, and melena neonatorum, may be associated with convulsions. But when this age is past, when the infections and injuries connected with birth no longer come under consideration, the clonic convulsions of functional nature become exceedingly rare.

To investigate the frequency of convulsions in the new-born D'Espine sent letters to the directors of various obstetrical clinics, who confirmed the great rarity of convulsions in the first days of life, but almost unanimously declared that clonic convulsive conditions occasionally occur *versus exitum* with *congenital debility* and in the absence of cerebral or intestinal infection. A number of these convulsive attacks, which are not always easy to explain, can probably be traced to septic infection, but another series of cases may depend upon a sort of autoinfection which in some instances can be referred to a deficient function of the parenchyma of the liver and kidneys.

Clonic convulsive attacks with loss of consciousness also occur during the *first weeks of life* as the result of severe *acute gastrointestinal diseases*, in which there is profuse loss of fluid, causing a deficiency of water in the tissues, especially of the brain. At this age severe inflammatory *pulmonary diseases* are not rarely associated with clonic spasmodic attacks.

The convulsions under consideration have been designated by Thiemich as *terminal*, probably for the reason that the appearance of this condition in early infancy in the course of the previously-mentioned diseases is of ominous import and frequently introduces the agonal stage. But in spite of the convulsive attacks it is not uncommon that life is maintained in very ill infants provided recourse is had to an energetic introduction of water by subcutaneous infusion.

The occurrence of convulsions *in the new-born children of eclamptic mothers* requires brief consideration. Without discussing the theory of *eclampsia gravidarum* it may be stated that, especially in the investigations of French authors (Moussous, Micholet, and Perret), changes in the kidneys and in the liver have been observed in these children quite similar to those in the eclamptic mother. There can be no doubt that the fetus may participate in all autointoxications from which the mother suffers, so that an explanation of the convulsive phenomena in the child of an eclamptic mother cannot be very difficult, even if such an occurrence were common.

**Prognosis.**—In regard to *prognosis*, the seriousness of the individual attack of simple convulsions is much more favorable than in true eclampsia. While it is no great rarity to see a child succumb to a spasm of the larynx combined with an eclamptic attack, simple convulsions as such, even in the severest diseases of childhood, with the exception of whooping-cough, are seldom the immediate cause of death. This does not exclude the fact that the underlying affection which was the cause of the convulsive attack may itself bring about the

fatal issue. However, experience teaches that in general the appearance of convulsions in the course of other affections is to be regarded as of unfavorable prognostic import only in infancy, and especially in the earliest period, and here the intestinal diseases are of more serious presage than diseases of other organs.

#### B. MYOTONIA OF THE NEW-BORN AND YOUNG INFANTS<sup>1</sup>

The external phenomena of the second group of occasional convulsions of children is to a certain extent in contrast to the class of convulsions just described. In the following forms of convulsions we are concerned with *persistent flexor spasms of the extremities* which are exclusively under the influence of *constitutional diseases* which damage the entire organism and occur only in earliest life. I described this peculiar spasm of the new-born and of young infants in 1900 in a special monograph on the "Myotonia of Nurslings." As these permanent convulsions are always dependent upon other manifest and easily diagnosticated affections of infancy, and, when life is retained, always disappear with the underlying disease, their inclusion in the group of occasional convulsions of children is well justified.

We will now turn to a brief consideration of these peculiar forms of spasm of early childhood.

In very sick infants, particularly those suffering, during the first weeks of life, from disease of the skin or of the intestines, from syphilis, or from sepsis, we frequently observe a peculiar *change of the muscle tonus in the extremities*. The limbs are constantly and spasmodically flexed and abducted, the hands are spasmodically contracted, forming a fist with included thumb or in a manner resembling tetany, and the toes, as a rule, are flexed spasmodically plantarward.

Prior to my first report there were but few references in literature to the occurrence of such persistent convulsions of the muscles of the extremities in babies, and these emanated from Bednár, v. Widerhofer, Epstein, Soltmann, Niemeyer, Henoeh, Strümpell, Baginsky, Czerny and Moser, and lastly from Zappert.

In the reports of some of the older authors the persistent contractures of the muscles of the extremities which are now under consideration have frequently been included in tetany. Such a classification was not surprising because at that time galvanic stimulation of the muscles and nerves had not been practised; otherwise the absence of electric hyperirritability would have indicated that these tonic convulsions of young nurslings are in no relation to tetany.

Niemeyer has proposed the name "*arthrogryposis*" for these contractures, and this nomenclature was in later times frequently employed for the genuine tetanic convulsions of infancy, although, as we shall learn later, they were altogether different from these permanent contractions. Henoeh and Strümpell are opposed to the idea of a similarity of these *persistent spasms* or "essential contractures," as they designated them, to tetany. Baginsky and Escherich

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<sup>1</sup> Not to be confounded with *myotonia congenita* or *Thomsen's disease*.



did not at first sharply differentiate these forms of spasm from tetany but have probably reached the conclusion ere now that between these permanent spasms of infancy and tetany there is no relationship. On the other hand, a young French author, Saint-Ange Roger, has recently attempted to include the permanent forms of contraction of earliest life among the "*formes frustes*" of tetany, in which he holds that the absence of hyperirritability of the nervous system is immaterial.

The essential criterion in the form of convulsion in question is the presence of symmetric, tonic flexor spasms of the extremities in children six to eight weeks of age, and the characteristic peculiarity that the contractures are *not intermittent*, like the genuine tetanic convulsions, but are *persistent*, and that neither galvanic nor mechanical hyperirritability of the motor nerves is present.

In order to understand the actual condition in tonic persistent contractures of the extremities of early infancy it must first be pointed out that even the normal attitude of the *new-born* is characterized by a certain *flexion hypertonia of the muscles of the extremities*, which gradually disappears in later months. In the new-born there is a physiologic tonic flexion of the elbow- and knee-joints, a flexion and adduction of the hip-joint, and a slight flexure position of the metacarpo-phalangeal and phalangeal joints of the upper extremities which is not present at a later period. With this the thumb is much more flexed than the fingers, and occasionally somewhat included so that the last phalanx is hidden. The hand of the normal infant, when at rest, usually appears as if forming a fist. This physiologic flexion of the extremities in the normal new-born may be readily overcome passively, but when the extremity is dropped it rapidly reappears. As a matter of fact, not only the flexors of the extremities but the entire musculature of the new-born is firmer and more rigid than that of older children.

Exceptions to this are found only in *myxedematous* and *mongoloid new-born* and young infants in whom the physiologic hypertonus of the muscles, for some remarkable reason, is absent and instead, quite an opposite condition, namely, a peculiar flaccidity, a kind of *hypotonia*, is noted. In my monograph upon myotonia of the new-born, previously quoted, I pointed out this circumstance, and particularly remarked that the position of the lower limbs, in children of this category, instead of being adducted and flexed at the thigh, present a complete *abduction and outward rotation* with a perfectly flaccid musculature.

In following the process of development of a normal child it will be found that this flexion hypertonia does not disappear in a few days; on the contrary, even under normal circumstances, this phenomenon may exist for a couple of months, become less marked in the third month, and disappear in the fourth or fifth month.

The peculiar muscular rigidity of the new-born, as also the immoderate flexion hypertonia and the tendency to persistent tonic muscular convulsions, is very readily understood when we consider, on the one hand, the state of irritability of the muscles and motor nerves at this age, described by Soltmann and

both Westphals, and on the other, the innervation of the voluntary muscles, explained by Kassowitz.

In my monograph upon myotonia of the new-born an attempt was made to base the peculiarities of the musculature in the first weeks of life simply upon the results of Soltmann's experiments, from which it appeared that the muscles of the new-born animal, even with an exceptionally slight frequency of stimulation conducted to the nervous apparatus, showed tonic contraction. Therefore, I considered that the slight external stimulations which under normal circumstances are conducted to the spinal cord, that is, to the motor nerve roots, would act much more intensely upon the muscles of the new-born than upon those of older individuals. This condition would be favored by the *absence of inhibition on the part of higher centers* which exists in the earliest epochs of life, so that an undiminished transmission of the impulses conducted from without to the motor anterior roots of the spinal cord was possible.

Nevertheless, it was incomprehensible why stimulations conducted from the center to the periphery gave rise *exclusively* to muscular *contraction* and never to *elongation*. In other words, the peculiar rigidity of the muscles of the new-born found an analogy to Soltmann's experiments in new-born animals, but its further nature remained altogether obscure. Only through Kassowitz's theory of muscular innervation, which shows that the impulses for relaxation, or elongation, of the muscles proceed in the inhibitive nerves, while those for shortening, or contraction, of the muscle are found in the stimulating tracts, is the peculiarity of the musculature of the new-born explained. That this physiologic muscle rigor is also in part associated with the incomplete development of the conduction tracts is obvious from the study of the muscles of *premature infants*, in whom the muscular rigidity and flexion hypertonia of the limbs are more distinct than in the normal child and also continue for a longer time.

Furthermore, it is obvious that the tendency to flexion in the new-born must be assumed to be a continuance of the physiologic intrauterine position, and that in the first extra-uterine months the congenital predominance of the flexor muscles over the extensors which exists throughout life is exaggerated.

From this it is easy to understand why, in severe disturbances of health which result in greater sensitiveness of the spinal cord and an increase of the stimulations which flow toward it, an exaggeration of this physiologic muscular rigidity may extend to permanent tonic flexure convulsions. The peculiar myogram of the new-born, which results from electric stimulation and resembles the contraction curve of an *exhausted muscle*, is readily understood when we consider the still deficient development of the medullary sheaths in the conduction tracts and motor nerves in the first days or weeks of life.

As a result of all of these circumstances the muscles of the new-born do not relax when numerous and intense impulses are conducted to them, therefore there is a condition of tonic contracture in which the flexors and adductors, which are already in a hypertonic condition, are principally involved.

Under the influence of psychic irritation even in normal nurslings the physiologic flexion of the limbs is transitorily increased to a spasmodic flexor

stiffening in the upper and lower extremities, whereby the hands are frequently doubled into a fist and the toes are flexed plantarward. But it would be incorrect to regard this condition, which occurs many times daily during crying and screaming, as pathologic or as a transitorial stage to tetany, although in older children the attitudes of intermittent tetanic attacks are not dissimilar. This phenomenon also can be readily explained in the sense of Kassowitz's theory of muscle innervation. Muscular rigidity of young infants in conditions of excitement is a reflex act and occurs in the earliest age without any apparent coöperation of inhibitive fibers, which, as Kassowitz has explained, are exclusively associated with elongation or relaxation of the muscles. Therefore the muscle-shortening action of the stimulating tracts is greater during the first weeks of life and muscle rigidity becomes noticeable even with slight reflex stimulation of the motor nerves. But the spontaneous movements of a normal nursling, with the peculiar flexor rigidity of its extremities, result without this stimulation and for this reason they appear somewhat helpless and marionette-like.

Between the physiologic condition of muscle tonus of the new-born, which finds expression in a mild flexion attitude of the extremities, and the tonic persistent flexor- and adduction spasms of early infancy there are numerous transitional stages.

In the first six or eight weeks of life the muscle tonus is influenced in the sense of a hypertonia by the most varied disturbances of health, but especially by those of *gastrointestinal nature* and the prolonged inflammatory or irritative conditions which originate from the skin. The first and most important evidence of a change in this respect is the appearance of the *fist phenomenon*, first described by me. If in these infants pressure is made upon the brachial plexus within the sulcus bicipitalis internus and the vessels are subjected to firm pressure by the finger upon the bones of the upper arm, or if the upper arm is encircled by an elastic band, a *spasmodic closure of the fist* is observed, as a rule simultaneously with the anemia of the hand from compression of the blood-vessels, but frequently somewhat later. The thumb is spasmodically flexed within the closed fingers, opposed or vertical to them; more rarely it is extended, completely rigid, between the first and second fingers.

In the affected child the contracture of the fist as a rule continues somewhat longer than the compression. Sometimes, and particularly in the milder cases, the fist phenomenon appears at the beginning of the compression of the upper arm but disappears spontaneously during the compression.

The fist phenomenon is probably an artificial flexor contraction of the hand and forearm, produced reflexly, which occurs so readily during the first weeks of life in sick children, because the innervation of the flexors at this early age prevails normally to a marked degree and, as was shown by the previously mentioned experiments of Soltmann, on account of the slight inhibitive action of higher centers, even weak stimulation at long intervals upon the motor nerves is sufficient to cause a tonic muscular contraction. The permanent contraction as such, however, is the consequence of a predominance of the innervation of



the myoplasma over that of the sarcoplasma, for in the earliest period of life weaker and slighter stimulations are conducted to the latter than to the fibrillary substance of the muscles.

It is of essential importance to note that the artificial development of the fist phenomenon *disappears almost entirely after the second month of life* and that after this epoch a tonic spasmodic phenomenon in the hand can be produced artificially under the influence of genuine tetany, although, as we shall note later, spasm of the hand in genuine tetany, artificially produced, does not differ materially in its external aspect from the fist phenomenon just described.

Trousseau's phenomenon of *true tetany* occurs only in older infants, and then notwithstanding the presence of reflex inhibitive influences from higher centers, because tetany is associated with decided hyperirritability of the peripheral nerves by which apparently the reflex inhibitive influences of the brain are paralyzed. Therefore there is no contradiction in the statement that in two neuro-muscular disturbances of infancy, which differ essentially in their nature, similar symptoms may be artificially produced.

Children in whom the fist phenomenon can be produced even in a condition of repose possess a more intense flexor hypertonia than is physiologic. The muscle substance of the flexors appears somewhat harder and the resistance to passive extension of the limbs is greater than under normal circumstances. Such children very frequently show a constant abnormal rigidity of their *entire musculature*. In more marked grades in which the tonus of the muscle is influenced, spastic positions of the extremities are noted, presenting the type of conspicuous flexor contractures, which may be quite properly designated *permanent spasms*. In this condition the position of the hand is commonly that of the fist phenomenon; more rarely it resembles the condition of tetany. In these persistent spasms the toes may present a claw-like plantar flexion. The flexors of the extremities are more prominent, rigid, and occasionally even of marble-like hardness. The joints are extended with difficulty, the fists can only be opened passively and with great exertion and are closed at once upon relaxation of the effort. The wrists are flexed palmarward *in toto*. Sometimes the entire hand is pronated and abducted as in tetany. Occasionally the fingers assume a paw-like position and the upper extremities a spastic condition which resembles the begging attitude of dogs.

Nevertheless, it would be a mistake to assume that under the above circumstances only the flexor muscles show a pathologic condition. Just as in the entire musculature of the normal new-born there is an increased tonus, so in these contractions there is a general pathologic rigidity, and only the functional preponderance of the flexors and adductors, which is normal, produces the flexure or adduction contraction of the limbs.

Most intensely involved in this hypertonia are the *flexor pollicis* and *opponens pollicis*. This is true of the physiologic as well as of the pathologic flexure hypertonia and also of the spontaneous permanent spasms and the fist phenomenon.

The pathologic tetanoid conditions which Escherich has termed "*pseudo-tetanus*," in so far as they concern nurslings during the first weeks of life, belong to the realm of myotonia. The condition is a spasmodic affection of all of the muscles of the body resulting from toxic influences, and therefore, in young infants, is only observed in severe sepsis, syphilis, or intestinal disease. On the other hand the pseudo-tetanic affections of Escherich which occur in later infancy belong partly to the realm of tetany and partly to genuine tetanus, and also to a certain extent are manifestations of hysteria.

It must be added that in the pseudo-tetanus of infancy tonic contractures of the extremities occur with the arms in the position of fencing and with opisthotonos and contraction of the neck, and that only one symptom which belongs to true tetanus is almost invariably absent, i. e., trismus or spasm of the jaw. But even these varieties of spasm have been noted in high-graded pseudo-tetanus.

There is no doubt in my mind that many of the cases of recovery from *tetanus neonatorum* which have been reported in literature were no more than severe grades of the myotonia under consideration, in which the muscles of the trunk were involved in addition to the muscles of the extremities. Such an erroneous view of pathologic conditions resembling tetanus is explained by the fact that severe forms of myotonia develop mainly in septic processes, and in particular in disease of the umbilicus, from which, as is well known, genuine tetanus may also originate.

It is important to know that the spastic conditions just described have been more or less persistent for days or weeks, although possibly with slight remissions, that there is entire absence of pain, that these convulsions never appear in paroxysms as in genuine tetany but that they develop gradually and, if the underlying affection has disappeared, as gradually ameliorate.

In regard to flexion of the hand and fingers, it must be stated that even in persistent spasm with convulsive closure of the fist, spontaneous movements of the fingers, although of slight excursus, may be observed, especially when severe reflex irritation (prick of a pin, pinching, and the like) is applied to the extremities. Further, that in the pathologic myotonia of nurslings there is apparently a diminution of the deep reflexes which may probably be referred to the difficulty in movement of the rigid extremities.

The myotonic spasms of the new-born and young infants are in no direct dependence upon febrile processes, although, according to the nature of the underlying affection, they may occur in febrile diseases. As previously mentioned, diseases with high fever are occasionally associated in the earliest epochs of life with convulsions, and I have also observed febrile pathologic processes in the new-born which began with initial convulsions and later were followed by permanent muscular spasms of the extremities.

Myotonia of nurslings is never a primary disease but is always dependent upon other disturbances of health. Very likely the muscular rigidity here under consideration is only the consequence of nutritive disturbance or of toxic influences upon the spinal cord, which manifests itself toward the periphery in

the motor end organs of the nerves of the muscles in the manner previously described.

The more severe the underlying affection and the longer its duration, the more intense are the tonic convulsions of the extremities. Thus we find the most marked grades of myotonia, with a convulsive picture resembling tetanus, in severe and prolonged diseases associated with a toxic condition of the central nervous system, as in gastrointestinal autointoxication, in severe cutaneous inflammation, burns, and in congenital syphilis, while in slight disturbances of health, as a rule, only the fist phenomenon is present without persistent spasm. If the underlying affection is relieved the myotonic spasm dependent



FIG. 11.—MYOTONIA SPASTICA PERSTANS  
EX ENTERITIDE CHRONICA. ATROPHY.

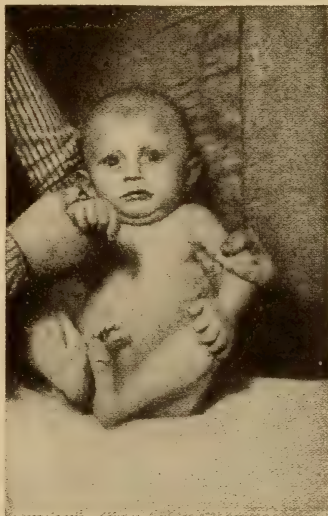


FIG. 12.—THE SAME CHILD THREE MONTHS  
LATER. TREATMENT IN THE DRESDEN  
HOME FOR INFANTS.

upon it also disappears. The illustrations (Figs. 11 and 12), which I owe to the kindness of Prof. Schlossmann of Dresden, depict the condition better than any description.

*Hereditary syphilis* plays a great rôle in the etiology of myotonia neonatorum. According to my observations almost all children affected with florid congenital syphilis in the first six to eight weeks show the spasms of myotonia, and for the most part the spontaneous form, more rarely the artificially-produced fist phenomenon. The dependence of the contractures upon the syphilitic influence of the central nervous system is clearly demonstrated by the prompt result of treatment, as a therapy which brings about an amelioration of the syphilitic symptoms also gradually relieves the convulsive phenomenon.

Myotonia of early infancy may be divided clinically into four groups:

I. *Myotonia physiologica neonatorum*: Slight rigidity of the flexors of the



extremities and a tendency to slight flexion of the fingers and toes in an otherwise normal child.

II. *Pathologic myotonia of the first grade*: The demonstration of the fist phenomenon and increased general flexor hypertonia during rest.

III. *Myotonia of the second grade (myotonia spastica perstans)*: Permanent tonic flexure spasms of the muscles of the extremities, particularly of the hands and feet (arthrogryposis, essential contracture of some authors), occurring in connection with autointoxication, severe intestinal and cutaneous diseases, hereditary syphilis, and burns.

IV. *Pseudo-tetanus*: Implication of the muscles of the trunk and perhaps also of the face in the myotonic processes; otherwise resembling the third group.

In the last three groups the fist phenomenon commonly occurs within the first two months of life (approximately). The spontaneous persistent spasms of myotonia, however, occur during the entire period of infancy, although after the first two months they are more rare.

The following differential factors are opposed to tetany: The contractures of myotonia are persistent, do not occur in paroxysms, are painless. They do not appear abruptly; on the contrary, the muscular rigidity develops gradually, corresponding to the causal affection. There is neither mechanical nor galvanic hyperirritability of the nerve trunks. The facial phenomenon is never observed. Laryngospasm and eclampsia are absent. Myotonia occurs principally in the first weeks of life; it is not dependent upon season, although it is more common in summer on account of the tendency to severe intestinal disturbance. There is no connection with rickets. Relapse does not occur after the exciting cause is removed. Finally, treatment with phosphorus, so effectual in the hyperirritability spasms of the rachitic, is without influence in myotonia.

## II. HYPERIRRITABILITY CONVULSIONS OF INFANCY

The second principal group of infantile convulsions are designated "*hyperirritability convulsions*" because in the intervals the children in question present disturbances in their psychic and nerve functions which we have briefly expressed by the term "*hyperirritability of the nervous sphere*." We must now explain what constitutes this hyperirritability of the nervous sphere and describe the convulsive picture and the period free from attacks.

The physiologic spasmophilia of early infancy, as Thiemich has quite properly emphasized, is not identical with the hyperirritability which is the basis of the variety of convulsions in question. On the contrary, we are here concerned with a *continued pathologic condition* of the central nervous apparatus, particularly of the *cerebral cortex*, which may also be demonstrated clinically in the interval free from attacks.

The decided tendency to convulsions which is present in the first years of life, even under normal circumstances, is decidedly increased under the influence of a persistent hyperirritability of the nervous sphere, as is readily understood, so that here, inversely to the previously described occasional convulsions, an intense disturbance of health is not necessary for the spasmodic attacks.

On the contrary, in these hypersensitive children very insignificant causes and even common occurrences are sufficient to produce an attack, which, in contrast with the harmless occasional spasms, may be of life-threatening importance.

Before entering into a detailed description of hyperirritability convulsions it must be remarked in regard to their external manifestation that a special form of *respiratory spasm* plays the most important part, whereby the clinical picture of this group of spasms differs from that of the occasional convulsions which are characterized by spastic attacks. The important form of spasm to be considered here is *spasm of the glottis*, to which, at this point, a few words must be devoted. In its mildest forms, as a "sighing" inspiration, or as a momentary arrest of respiration during crying, which occur daily in many children, it presents one of the most essential symptoms of hypersensitiveness. However, if the child who shows this peculiarity is closely examined it will be found that other symptoms denoting a hypersensitive nervous sphere are never lacking. Such cases are characterized throughout by an abnormal irritability of the psychic condition, by timidity, anger, often by insomnia and a special sensibility. Briefly, there is always a special psychic irritability which is a predominant characteristic.

Many infants betray their hypersensitiveness by starting at the slightest noise, panting with any excitement, showing anger if their will is crossed. These respiratory spasms arise under quite ordinary circumstances from which they accidentally become excited. The attacks are often induced by anger at being taken from the breast before the child has finished nursing. The reason why these symptoms of pathologic irritability have frequently been incorrectly estimated is explained by the fact that in practice they are regarded as of secondary importance, and that gasping during crying, a daily occurrence in many children, has not been appreciated to a proper extent.

These symptoms of an exaggerated irritability of the nervous sphere in infancy commonly arise between the sixth and thirtieth months, and the first circumstances which draw attention to the hyperirritability in question are the gasping of the child while crying and the crowing inspiration in psychical agitation. In addition to these predominant psychical phenomena very frequently there are signs of hyperirritability of the peripheral nervous system which are referred to mechanical and galvanic influences. Thus, in many children of this category *Chvostek's facial phenomenon* and an *increased galvanic irritability* may be demonstrated which, when they occur after infancy, are characteristic of a quite definite functional neurosis—*tetany*. These circumstances have led a number of authors to regard such convulsions of children as tetany. In these cases Thiemich has attached special value to the *galvanic hyperirritability* of the nerve and when increased galvanic irritability is present during the period free from attacks the child is declared by this author to be in a "*tetanoid condition*." Accordingly he differentiates the convulsions associated with galvanic hyperirritability from those in which such a condition cannot be demonstrated, and includes the spasmodic attacks of the first cate-

gory in the domain of *tetany*. For this he employed a particular factor, as we shall hear later, namely, the reaction to CaOC, to differentiate between the tetanoid and non-tetanoid convulsions of children.

Although refraining from a discussion of these conditions until later, it must be remarked at this point that a positive criterion for the forms of convulsion associated with respiratory spasm and psychical hypersensitiveness is not to be found in the presence or absence of galvanic hyperirritability. On the contrary, according to my judgment, the galvanic hyperirritability of these children is only an accompanying phenomenon of the increased irritability of the entire nervous sphere which is the pathognomonic sign of the general disturbance to which these convulsive conditions are due.

For example, we cannot admit that the spasm of the glottis has a different significance when the child shows a galvanic hyperirritability than if it should present a milder reaction, especially as definite normal values for the galvanic irritability in the individual months of life do not exist. Neither the clinical convulsive picture nor the psychic behavior of these children in the intervals between the attacks is changed in any manner by the presence or absence of galvanic hyperirritability.

However, it must be stated at the outset that among the spasms of early infancy associated with general hypersensitiveness of the nervous sphere there is one form which coincides in all respects with the *tetany* of adults, so that we must differentiate the simple hyperirritability convulsions of children, immaterial whether they be associated with galvanic hyperirritability, from true tetany in which the characteristic spontaneous or artificially-produced carpopedal contractions are present in addition to the demonstrable mechanical and galvanic hyperirritability.

There could be no objection to the designation of the hyperirritability convulsions of children as "*infantile tetany*," as was done by Escherich and his pupil Loos, provided it were borne in mind that between the genuine tetany of later years and the simple hyperirritability convulsions of children there is a vast clinical difference. To prevent confusion it would be necessary to differentiate between "*infantile tetany*" and "*genuine tetany of children*." I believe it to be more serviceable to make a sharp differentiation and only to specify those convulsions of children as "*tetany*" which agree in every particular with the tetanic spasms of adults. Every other form of convulsion should be designated by its clinical name such as spasm of the glottis, eclampsia, tetany, etc.

In regard to the clinical pictures of the convulsions which here come into question, we must differentiate four principal forms:

1. *Respiratory spasms.*
2. *Eclampsia.*
3. *Genuine tetany.*
4. *Nodding spasms of children.*

These are the forms of convulsion which even to-day are often designated idiopathic or essential convulsions of childhood because upon superficial investigation they readily appear to be a substantive disease. We shall first



describe the clinical symptoms of the individual forms of hyperirritability spasm and then consider the pathogenesis in general.

#### A. RESPIRATORY SPASMS OF CHILDREN

According to the phase of respiration in which the respiratory spasms occur it is necessary to subdivide them into two groups, the *inspiratory* and the *expiratory* forms. Until recently all respiratory convulsions of infancy were included under the collective term "spasm of the glottis" (laryngospasm, laryngismus stridulus). But, as Kassowitz quite correctly asserts, not all of the spasms of the glottis, so-called in pediatrics, are actually associated with a spasmodic activity of the constrictor muscles of the glottis. On the contrary, the respiratory convulsion very frequently manifests itself by an *expiratory spasm*, in which after a few rapid successive expirations, there is sudden complete arrest of breathing, "*expiratory apnea*."

*Spasm of the glottis*, in the restricted sense of the term, is a true *inspiratory spasm* with a sudden spasmodic contraction of the glottis which may be recognized by a sudden crowing, gasping or sighing breath—the so-called "choking of children"—instead of the normal, quiet inspiration. Some attacks consist exclusively of these inspiratory spasms; again, there are attacks in which no spasmodic inspirations appear but the child apparently remains in the expiratory stage of respiration, popularly called "arrest of breathing." These two forms may be combined, i. e., after an unusually protracted, long-drawn-out arrest of breathing in an expiratory position no free inhalation but a laryngospastic, noisy inspiration results. Inversely, it may happen that an inspiratory spasm of the glottis introduces the attack, after which the respiration again becomes free, but subsequently, following several forcible and noisy inspirations there is a sudden expiratory arrest and a long time elapses before free inspiration again, if at all, recurs. Both forms of convulsion, provided they appear independently of each other, may lead to high-graded asphyxia and terminate fatally. Much more serious than simple inspiratory spasm of the glottis, as was shown by Kassowitz, are the attacks of expiratory apnea which are often the immediate cause of death.

In very mild attacks, lasting but a few seconds, consciousness is not necessarily lost, but the severer attacks, particularly of expiratory apnea, begin immediately with unconsciousness.

The more common variety of respiratory spasm is the spasm of the glottis. Of all the convulsions of children associated with hyperirritability this, according to my investigations, is the most frequent and relatively the earliest. There is quite a series of cases in which it has occurred substantively, i. e., without association with other forms of convulsion except those mild attacks of rigidity which occur during the course of the paroxysm and last but a few seconds. In many cases, however, it is an initial or partial phenomenon of a well-defined *eclamptic attack*, associated with clonic-tonic contractions of the muscles of the extremities and loss of consciousness. Spasm of the glottis in untreated cases is usually a periodically recurrent hyperkinesia which continues for sev-

eral months and even for years. Not rarely these attacks occur daily, sometimes repeatedly during the same day or as often as the child is subject to some psychical irritation. In untreated cases this condition may last from the sixth to the twenty-fourth month of life and even longer.

The ordinary mild form of laryngospasm is characterized by a shrill-toned inspiration and a brief arrest of respiration. Naturally the sound is only the expression of the spasmodic adduction of the vocal cords which, upon the attempt to inspire air between them, results in noisy vibrations.

The muscles herein involved are those innervated by the recurrent laryngeal and pneumogastric nerves, namely, the thyro-arytenoids, the crico-arytenoids, and the transverse arytenoids.

In the milder cases nothing is heard but a sighing, crowing, or piping sound which is associated with a fruitless attempt at inspiration. Prior to this, for an exceedingly brief time, the child has been *pale*, but after the spasm has lasted a few seconds it will be observed that this initial pallor gives way to slight *cyanosis*. Ordinarily the extremities are extended and become somewhat rigid simultaneously with the crowing inspiration.

After the inspiratory arrest has lasted a few seconds the spasm terminates in a *deep inspiration* and the attack is over. In the majority of these mild cases of laryngospasm the relatives of the child become accustomed to these paroxysms, provided only the crowing and piping sounds are heard, and attach no importance to them.

In severe attacks the condition is different. Very frequently they begin with an *expiratory arrest of breathing*, quite without the crowing attempts at inspiration. In connection with some external irritation such as hawking, an examination of the larynx with a spatula, sudden chilling or pain, or some psychical irritation (crying, screaming, awakening from sleep), a quite unexpected and lightning-like expiratory arrest of respiration appears. The child rolls the eyes and presents tonic extensor spasms of the extremities, very frequently with tonic contractions of the hands and of the fingers. Sometimes there is also a simultaneous tonic contracture of the knee. The abdominal muscles and the diaphragm show a tonic contraction, and after this tonic spasmodic condition has lasted one or two minutes twitching in the extremities takes place. With this there is a shrill, usually crowing, sound which indicates the termination of the expiratory spasm and the re-entrance or, better, the spasmodic inhalation of air through the glottis which is but slightly opened. This crowing may be repeated several times at brief intervals, but finally disappears with the relaxation of the laryngeal muscles and a normal passage of air through the glottis.

After these severe attacks the child is exhausted and often falls asleep. Occasionally a decided pallor of the face remains for several hours. Not always, however, do these attacks pass off in such a harmless manner. There are cases in which the child suffocates in a condition of expiratory apnea. The crowing inspiration does not occur, as in consequence of the tonic spasm of the laryngeal muscles not an atom of air can pass through the glottis. This form

of respiratory convulsion in particular, which begins directly with tonic expiratory spasm, whereby the diaphragm and the muscles of respiration retain a spasmodic expiratory position and the muscles of the larynx are firmly contracted, is the one in which the eclamptic attacks terminate fatally and which Kassowitz has so classically described.

Tracheotomy and intubation are useless in this form of expiratory apnea. The condition is due to a central hindrance of inspiration, to a complete fixation of the entire respiratory musculature in the stage of expiration, which remains even when the communication of the larger respiratory passages with the external air becomes free.

#### B. ECLAMPSIA OF CHILDHOOD

By infantile eclampsia in its restricted sense we understand the periodic occurrence of tonic and clonic convulsions of the external musculature during the first three years of life in children who are affected by permanent hyper-irritability of the nervous sphere. There is no definite type of eclamptic attack. In their intensity and duration the convulsions are subjected to enormous variation. Occasionally a slight contraction of the muscles of the face, associated with rolling of the eyes, has the same significance symptomatically as an attack, lasting for hours, which begins with tonic rigidity of the extremities and passes into severe convulsions of the entire musculature of the body.

Severe attacks, as a rule, present the following clinical picture:

The scene opens almost invariably with a spasm of the larynx, mostly inspiratory, but occasionally of expiratory type, combined with general muscular rigidity. This tonic stage with arrest of respiration seldom lasts longer than half a minute; the respiratory spasm then ceases and is followed by a clonic contraction of the muscles of the extremities of incalculable duration. Consciousness is always lost. The eyes are staring, the corneal and pupillary reflexes are absent, the glance is rigid and directed into space; very frequently, however, the pupils are not visible, as the eyeball is rolled so far upward that the cornea is completely covered by the upper lid. The clonic and tonic spasms of the facial muscles are particularly conspicuous. Upon close investigation it is found that the jaws are pressed tightly together; occasionally there is frothing at the mouth and spasmodic lateral movements of the lower jaw are executed with tonically contracted masseter muscles, as in genuine epileptic attacks. The respiration is labored and noisy, very often irregular; the pulse is rapid and arrhythmic. Frequently there is spontaneous evacuation of urine and feces. If the condition occurs in nurslings with an open frontal fontanelle this structure is found markedly protruded and tense. Severe attacks may cause immediate death. As a rule, however, the convulsive picture gradually resolves itself into an amelioration of the clonic and tonic contractions and the child falls into a deep sleep lasting several hours. Subsequent to the attack the patient is pallid and exhausted, and as a rule this lassitude continues during the entire succeeding day. Occasionally the post-eclamptic sleep is interrupted by a renewed attack.



It is a disputed question whether or not an attack may last for hours. Many authors believe that the individual paroxysm is brief, not longer than a minute, and that prolonged attacks are merely the grouping of numerous individual paroxysms which pass one into another.

The first symptom is usually a change in the facial expression. Particularly conspicuous in children, and especially in nurslings, is a wrinkling of the forehead, denoting a spasm of the muscles of the head and face. A fish-like contraction of the mouth preceding these convulsions and a change of countenance, a staring and expressionless face, indicate that very often the muscles of expression are involved earlier in the spasmodic condition than the muscles of the extremities.

During the attack the skin is very often decidedly *cyanotic*, but this does not occur at the onset of the paroxysm; the attack is commonly introduced by *marked pallor* of the face, which does not give place to cyanosis until the appearance of respiratory spasm and arrest of expiration. The respiration very often does not suffer solely through spasm of the glottis and apneic attacks, which are only of brief duration and may recur during one and the same eclamptic seizure; on the contrary, difficult, stertorous respiration persists throughout the attack which proves that there is often tonic spasm of the entire respiratory musculature.

In my opinion the most important criterion of genuine eclampsia is the *presence of laryngeal spasm* prior to or during the eclamptic seizures, or in the interval between them, immaterial whether mechanical or galvanic hyperirritability is present in the peripheral nerves.

A few words must be devoted to the *condition of the extremities* during the eclamptic paroxysm.

At the onset of the attack the upper extremities are almost always in rigid flexion contracture. The upper arm is held tightly against the trunk, the hands with enclosed thumb are rounded into a fist, and also flexed. But general extension rigidity of the large joints may also occur at the beginning of the attack, and exceptionally I have noted that the paroxysm begins with a crowing, laryngospastic inspiration and extensor rigidity of the limbs, with the head fixed posteriorly, and that flexor contracture takes the place of extensor rigidity only upon relaxation of the glottis. The lower extremities, apart from contraction, are usually in an equinus position—the toes are spasmodically flexed plantarward. Occasionally, quite similar to the condition in the hands, the halluces of the lower extremities are bent under the second and third toes. Very often a tonic spasm of the extremities alternates with clonic contraction, which as a rule occurs rhythmically—simultaneously in the upper and lower limbs and in the head, I might say, in a regular tempo. Opisthotonos during the entire duration of the spasm is common. When clonic contractions appear in the extremities they are usually associated with a retraction of the head in the same rhythm. Occasionally the contractions begin unilaterally or are developed more markedly upon one side of the body than upon the other.

## C. TETANY OF CHILDREN

There is a spasmodic clinical picture of early childhood which coincides in all respects with the pathologic picture of the tetany of adults: tonic, intermittent convulsions of the extremities with peculiar rigidity of the fingers and eventually also of the toes, retained consciousness, and increased mechanical and galvanic irritability of the peripheral nerves during the intervals free from attacks.<sup>1</sup>

The appearance of this disease in infancy was first reported by J. Clarke (1817) and Tonnelé (1830). Later reports were issued by Steinheim (1830) and by Dance (1831). The name "tetany" was used for the first time by Trousseau's pupil, Corvisart, in the year 1832. It is not certain whether the cases which were at that time regarded as tetany of infants would be so designated to-day. Undoubtedly some of them should be included in the group of simple permanent contractions and therefore cannot be included among the "hyperirritability convulsions."

In infancy as well as in later life we speak of a *manifest* and a *latent tetany*. Regarding the conception of manifest tetany of infants there is unanimity. Tetany is manifest when the tonic spasms of the extremities, soon to be described, arise *spontaneously*; tetany is to be regarded as latent if certain phenomena of an *increased irritability of the nervous system* are present during the intervals free from attack, or even with complete absence of spontaneous spasm of the extremities.

The only disputed question is, which symptoms of hyperirritability exhaust the conception of *latent tetany*, that is to say, which are sufficient for a diagnosis of this condition.

We will first relate all of the symptoms of infantile tetany which have become clinically familiar and will then discuss their value in the diagnosis of latent tetany.

First in order are the *spasmodic symptoms*, and here we must differentiate between the *spontaneous* and *artificially-produced* spasms of the extremities. The latter, the so-called *Trousseau's phenomenon*, is a characteristic rigidity of the extremity which may be produced by constriction of the upper arm by means of an elastic ligature, or by pressure of the hand exerted upon the nerve trunks situated in the sulcus bicipitalis, for a period varying from thirty seconds to three minutes. During this artificially-produced tonic spasm the same condition may be recognized in the upper extremities, as is the case in the spontaneous spasms of tetany. The muscles of the hand and forearm are the most intensely affected, and particularly those supplied by the *ulnar nerve*. This causes a position of the hand which is noted in electric stimulation of the ulnar nerve: *flexion of the fingers at the metacarpo-phalangeal joints, with extension of the interphalangeal articulations and abduction of the thumb*—the so-called "*obstetric hand*." This is the typical position of tetany, but in severe cases there may be a more marked flexion of the phalangeal joints and simultaneously also of the entire hand.

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<sup>1</sup>See volume on "Diseases of the Nervous System," p. 907.

The claw-like plantar flexion may also appear in the lower extremities in severe spontaneous tetany, but I have never succeeded, by constriction of the lower extremities, in bringing about an artificial phenomenon, in children affected by tetany, analogous to Trousseau's phenomenon in the upper extremity.

In this connection it must be stated that only the artificially-produced spasmodic condition of the hand with the characteristic rigidity of the fingers, just mentioned, in the form of the "*obstetric hand*" or the "*writing position*," can be regarded as *Trousseau's phenomenon*. A very decided resistance must be present when attempts are made to produce this spasm passively. A simple phalangeal flexion upon compression of the extremities occurs in very many quite normal children and is of no diagnostic importance. That the spasmodic closure of the fist in the employment of Trousseau's experiment during the first weeks of life is no proof of tetany has already been stated.

In the cases which are characterized by spontaneous spasm Trousseau's phenomenon is developed readily, and especially so immediately after the cessation of a spontaneous attack. It has also been repeatedly reported to me that in the manifest tetany of children the production of Trousseau's sign has given rise to prolonged spontaneous attacks. Nor must it be overlooked that many hypersensitive children under the influence of Trousseau's experiment are seized with laryngospastic and eclamptic attacks, immaterial whether the phenomenon is actually elicited.

Errors are very liable to arise in determining whether the flexion of the phalanges with included thumb, which follows constriction of the upper arm, is to be regarded as Trousseau's sign. In some children a kind of resistant movement, a flexion of the fingers, occurs which resembles this phenomenon. If this flexion disappears of itself in the course of a few seconds or upon passive opening of the hand, it is not the contracture of tetany. Errors, however, can also occur by making too short a pressure or constriction. The affected hand must be completely emptied of blood and the constriction must have lasted at least two minutes, and only under these conditions does a negative result indicate that Trousseau's sign cannot be elicited.

Genuine tetanic spasms of children are associated with a definite age. They are rarely present before the first year of life and seldom after the close of the fourth. Of the external muscles only those of the extremities are involved in the attack. They are *in toto* firmer and more rigid than normal and the limbs are fixed in a slight flexion contracture. The muscles of the trunk remain normal. The most important spasmodic symptom, however, is the peculiar *position of the hand*, already described. During the attack a similar rigidity may occur in the toes as in the fingers.

The genuine tetanic spasms are never associated with *disturbance in consciousness*, except perhaps when they are introduced by a laryngospastic attack, which gives rise to unconsciousness but lasts only a very brief time. This condition disappears immediately upon the resumption of a normal respiratory condition. The respiratory convulsions play a much more insignificant rôle in



genuine tetany than in eclampsia and never have the threatening character of eclamptic conditions. There is no record of a child having succumbed to a tetanic contracture, and during its continuance the patient never appears to be an intense sufferer. In many children the contractures probably cause pain, as is obvious from certain resistant movements combined with crying upon attempts to relax the contracture passively.

In the spontaneous attacks of tetany the causative factor cannot always be demonstrated definitely in the form of some psychical irritation, as is the case in simple hyperirritability convulsions. In many instances the spasm of the extremities does not occur with lightning-like suddenness as in laryngospasm and eclampsia. Although the first symptom of the convulsion, the phalangeal rigidity, appears suddenly, the stiffening of the remaining musculature is usually gradual, and does not involve the entire musculature of the extremities until later. Genuine tetany always lasts longer, rarely less than fifteen minutes, and attacks continued for several hours with remissions in the intensity of the rigidity have frequently been reported. The spasm of genuine tetany of infants becomes gradually more and more intense and remains, often for hours, at a fixed height, and its disappearance, as a rule, is just as gradual.

It has always been my impression that the spasm of the glottis is quite a secondary condition in genuine tetany, and in fact is only a proof that here also a general hyperirritability prevails. The tetanic spasms themselves may last for hours without any evidence of respiratory spasm whatsoever. Sometimes the attack begins with laryngospasm, i. e., with a single, piping, spasmodic respiration, which does not usually recur during the entire duration of the contracture of the extremities. These genuine tetanic spasms, which must be differentiated from eclampsia on account of the absence of clonic muscular contractions, cyanosis, and unconsciousness, are relatively rare. In my experience they only appear in a cumulative form—*epidemically*. Thus in Vienna we had an epidemic of tetany in the spring months of the years 1899 and 1900; since then the frequency of genuine tetany in our hospital has constantly decreased so that in recent years I have seen scarcely more than two actual cases of tetany in children in any year. As a matter of fact genuine tetany is strictly associated with season. In Vienna it appears only in the late winter and in the spring months, reaching its acme in April; during the summer it is almost entirely absent.

Children who suffer from spontaneous genuine tetany always present an increased mechanical and galvanic irritability of the nerves during the periods free from attack and constantly show *Trousseau's phenomenon*, as well as the *Thiemich-Mann symptom*—a specially qualified galvanic hyperirritability which will be considered later.

I must caution against designating eclamptic attacks with rigidity of the limbs and loss of consciousness as tetany. What Elsässer has described as "*tetanus apnoicus infantum*" is not tetany but a transitory general muscular rigidity belonging to eclampsia which is accompanied by arrest of respiration and profound unconsciousness. Here quite a different spasmodic condition

from that of tetany predominates. The muscles of the extremities are extended rigidly, while the hands are rounded into a fist which cannot be opened; in addition the head is spasmodically fixed posteriorly, the face is blue, there is frothing at the mouth, and the muscles of the trunk are in a state of tonic spasm. As a rule this tonic stage is succeeded by a clonic contraction of the muscles of the face and trunk, which is a symptom foreign to tetany. The condition is due to a cortical irritation, while the irritative sphere in genuine tetany is predominantly infracortical, that is, of spinal origin.

A symptom that is never absent in the genuine tetany of infancy is the *mechanical hyperirritability* of the motor nerves. In all children who are subject to tetany this hyperirritability can be demonstrated in the facial nerve. To produce this facial phenomenon, which was discovered by Chvostek in 1876, and named for him, palpation of the pre-auricular portion of the cheek is necessary. When the hyperirritability is decided, a simple stroking of the cheek with the finger or with the shaft of a percussion hammer produces a lightning-like contraction in this muscular region of the face.

The facial phenomenon of tetany is in general more markedly developed in infancy than in later life. Neither in manifest nor in latent tetany is it absent. It is very frequently observed in children who are free from tetany and is especially common in those who suffer from a general hypersensitiveness of the nervous sphere. Accordingly, it is a common symptom in rachitic children who are subject to respiratory spasm.

The facial phenomenon is the expression of an increased irritability of the nerves of the face. There are hypersensitive children in whom a slight touch of the cheek with the finger is sufficient to produce an intense lightning-like contraction of all of the facial muscles upon the side in question. This condition can no longer be recognized as a symptom of tetany exclusively even when it is markedly developed. In this I am opposed to Hauser, Kraushaar, Ganghofner and Thiemich, who believe that a very intense result of palpation of the *ansa facialis* is a positive sign of latent tetany.

The *mouth phenomenon*, first described by Loos and Escherich, is to be considered in a manner similar to the facial phenomenon. Loos understands by this a lightning-like contraction of the angle of the mouth upon the side opposite to that of the palpated facial nerve, Escherich a contraction of the upper lip in sleeping children after percussion in the course of the constrictor muscle of the mouth, whereby the lips are protruded as in the act of whistling, Thiemich a rapid contraction of the orbicularis oris in children while awake when some point upon the upper lip is irritated mechanically (fish-mouth). It is evident that these three phenomena are only the expressions of a special hyperirritability of the facial nerve which innervates the orbicularis oris. In my opinion they are of no positive diagnostic value in infantile tetany, as they may also be produced in normal children who possess over-sensitive nerves of the face.

Less common and distinct is the hyperirritability of other nerve trunks. It may be recognized in the extremities by a lightning-like contraction of the

muscles supplied by the nerve in question when the nerve trunks are tapped with a percussion hammer. This may be tested most readily by percussion of the radial nerve where it curves in the lower third of the upper arm, and in the peroneal nerve which is stimulated externally from the head of the fibula by tapping lightly with the percussion hammer.

Lightning-like dorsal flexion of the hand results from mechanical irritation of the radial nerve; abduction and slight dorsal flexion of the foot by percussion of the peroneal nerve; but that these phenomena also occur in patients free of tetany and even in entirely normal children is even more true than of the facial phenomenon.

Of great importance in the latent and in the manifest tetany of infancy, just as in later life, are the changes of *electric contractility*. As was clearly shown by Kussmaul in 1872, and by Erb in 1874 and 1879, there is an *increased irritability of the motor nerves to both currents*. But faradic hyperirritability plays only a secondary rôle; the principal part, particularly in children, is assumed by the changes of *galvanic contractility*. We shall concern ourselves exclusively with the latter.

The *electric examination* should be undertaken in early infancy, as in older individuals, with Stintzing's normal electrode and without anesthesia, an electrode of 50 c. cm. serving as the negative pole. To gain uniform results for comparison it is very serviceable to employ the products of investigation of the median nerve, which is stimulated at the distal end of the sulcus bicipitalis internus. In regard to the *normal contractile relations* Mann has determined, analogous to the findings of C. and A. Westphal, that the sensitiveness of the motor nerve is less until about the eighth week of life than later, and that from this period to the end of the second year great variations occur which are not parallel to normal relations at the same age.

Nevertheless the contractility of advanced age is not reached in the first years of life; on the contrary, the values are always greater than in adults. Mann, who examined 13 children under eight weeks of age and 43 older children with the galvanic current, found that the normal value for CaCC of the median nerve in normal children from the third to the thirtieth month is almost invariably between 0.7 and 2.0 milliampères, while in adults, according to Stintzing, the corresponding value is 0.3 to 1.5 Ma.

Of great influence upon the contractility, according to Mann, is the nutritive condition of the child, for with a profuse adipose tissue stronger currents are necessary to produce a minimal contraction than with a thin cutaneous covering.

As Escherich determined, the values for CaCC which are characteristic of later life are subject to special variations in the first years and Thiemich has since stated that they are not decisive in all cases for the diagnosis of tetany.

Thiemich has rendered valuable service by demonstrating that in the CaOC a much more certain criterion exists for determining the presence of galvanic hyperirritability than was formerly the case in testing with CaCC.

In the tetany of adults the production of CaCC and of CaCT with relatively



mild currents is well known. These conditions were also studied in the great nerve trunks in small children by Escherich, Ganghofner, Hauser, Thiemich, and Mann. In the main the first three authors found similar anomalies of contractility in infantile tetany, while Thiemich and Mann were able to demonstrate that the early appearance of CaCT is not so important; on the contrary that the most important deviation from the usual contractile condition could be determined by CaOC. According to these authors a medium value under *normal* conditions for the last-named phase of contraction of the median nerve is 8.22 Ma. In latent tetany this average is diminished to 2.23 Ma. and in manifest tetany to 1.94 Ma.

As a limit between the CaOC of normal conditions and of tetany Thiemich and Mann have given the figure of 5 Ma. *Values below 5 Ma. belong exclusively to tetany, values above 5 Ma. are always normal.* Thiemich has gone further and upon the basis of this limitation in electric contractility of the motor nerves has attempted to divide the spasmodic phenomena of early infancy into two groups, whereby CaOC *below* 5 Ma. indicates a *tetanoid* condition, that is, is characteristic of latent tetany, while figures *over* 5 Ma. for CaOC indicate normal contraction of the peripheral nerves.

Ganghofner has failed repeatedly to demonstrate the figures given by Thiemich for CaOC in genuine tetany and is therefore not inclined to ascribe to them the diagnostic importance which is given by Thiemich. In fact he denies that the galvanic hyperirritability is of special significance in the diagnosis of latent tetany and believes that a sufficient support for the diagnosis of latent tetany is given by the presence of an especially increased mechanical irritability.

The values obtained by Escherich in testing the *nerves of the face* for CaCT in tetanic children under anesthesia cannot be utilized in practice because it is not always possible to make a galvano-diagnostic examination under anesthesia and the employment of stronger currents without narcosis causes an active tetanic spasm of the muscles of the face in consequence of the pain, which may simulate CaCT. However, it is necessary to emphasize that Escherich, aided by v. Wagner, was the first to demonstrate a measurable increase of galvanic contractility in infantile tetany by means of CaCT, that in the affected children this reaction appears with 5.2 and even with 1 Ma., while in normal children CaCT cannot be produced with currents of 20 Ma. and more. Escherich and Hauser assume a pathologic increase of electric contractility for CaCC when this occurs in the peroneal and ulnar nerves under less than 0.3 Ma., and in the facial nerve under 1 Ma.

In this connection two symptoms of tetany, recently described by Peters, must be mentioned, of which one, the "*jumping-jack phenomenon*," presents a special variation of electric hyperirritability, and the second consists of *increased values for pressure and amount of the cerebrospinal fluid in lumbar puncture.*

The first-named symptom is described as follows: Upon galvanization of the spinal cord at the cervical and lumbar enlargements, with the cathode upon

the vertebral column, the anode upon the breast, severe and rapid contractions appear in the upper and lower extremities which actively resemble the movements of a jumping-jack. This muscle play occurs in infantile tetany with 3 to 4 Ma., while in normal children two or three times the amount of current is necessary to produce the condition.

Hoffmann also assumed the presence of a reflex hyperirritability and a genuine *hyperirritability of the sensory nerves* in tetany in addition to the legitimate symptom triad which has already been mentioned. As a sign of this sensory hyperirritability the appearance of Trousseau's contracture following cutaneous irritation, for example, after the prick of a needle, after raising a fold of the skin, or after percussion on the vertebral column, has been reported. As for myself, however, I have not succeeded in producing Trousseau's contraction in infantile tetany otherwise than by constricting the upper arm.

The symptom triad—spontaneous or artificially-produced tonic spasms of the extremities, increased mechanical and galvanic contractility (Trousseau's, Chvostek's, and Erb's phenomena)—is characteristic of tetany at all periods of life. Through the investigations of Loos and Escherich in the ninth decade of the last century the teaching of tetany of children has included a relationship of that affection with *spasm of the glottis* and *idiopathic eclampsia*. These authors found that many children who suffered from such forms of spasm showed symptoms of tetany, namely, an increased mechanical and to some extent also an increased galvanic irritability of the peripheral nerves. They accordingly declared laryngospasm, and eclampsia occurring with laryngospasm, to be tetany, but generalized so far as to regard laryngospasm only as a part phenomenon of infantile tetany. Loos, in contrast to Kassowitz, at first denied all connection of these spasmodic conditions with rachitis.

The priority of discovery of the association of certain cases of laryngospasm with tetanic symptoms belongs to the London physician Gay, who, in 1890, published in the journal "*Brain*" an article upon spasm of the glottis. Gay reported that he found the facial phenomenon in 64 children, of whom 47 suffered with spasm of the glottis, 9 with general convulsions, 7 with genuine tetany, and 1 with *spasmus nutans*. Of these 64 children 54 had positive and 4 questionable symptoms of rickets. He therefore arrived at the conclusion that laryngospasm and tetany bore a certain relation to each other and were in some manner associated with rachitis.

The opinion of Loos and Escherich that laryngospasm is always a partial phenomenon of infantile tetany was rejected by most authors who afterward concerned themselves with this question, since it was determined by many that laryngospasm occurred quite frequently without other symptoms of tetany.

Upon comparison of various statistics regarding spasm of the glottis Kirchgässer found that this condition is almost twice as common as tetany and that it is associated with latent symptoms of tetany in barely 25 per cent. of the cases.

The exceedingly comprehensive compilation of the reports of different authors relative to spasm of the glottis and tetany, published by d'Espine in



1902, shows that the former condition is almost always much more common than tetany and that the genuine tetany corresponding to the affection in adults is almost unknown in certain regions, for example, in Geneva, Turin, Toulouse, and Munich. Of special interest are the conditions found in Geneva, concerning which d'Espine from his own observation reports that among 5447 children observed in his dispensary in a decade he has seen only 2 cases of actual tetany, 54 cases of eclampsia, and 20 cases of spasm of the glottis.

In the years 1899 to 1901 I carefully examined 82 cases of spasm of the glottis for symptoms of tetany and found that only 17 cases, therefore about one-fifth, were associated with genuine tetany in our sense of the term.

An article upon infantile tetany which Boral published in 1893, including 121 cases of spasm of the glottis from my hospital, showed that only in 13 were there symptoms of well-marked tetany, either spontaneous spasms or Trousseau's phenomenon. In addition to these the facial phenomenon was quite commonly observed in laryngospastic children. Altogether Boral found the facial phenomenon in 169 cases, frequently with laryngospasm but often independently of it and of other symptoms of tetany. In 24 cases Trousseau's phenomenon could be demonstrated simultaneously and in 14 of these there were respiratory spasms, in 21 the facial phenomenon.

The previously mentioned cases in my department, as well as those reported by Boral and those collected by myself for d'Espine's report at the Congress at Toulouse (1902), were not subjected to a thorough galvano-diagnostic test. Since Thiernich's report Dr. A. Schüller and myself examined many children from the neurological department of our hospital, who suffered from laryngospasm and eclampsia and presented the facial phenomenon, with the galvanic current, but were not always able to arrive at the figures for CaOC which were given as characteristic of latent tetany by Thiernich. This shows that mechanical and electrical hyperirritability of infants is by no means always the same.

Japha, who investigated more than 150 cases of laryngospasm in H. Neumann's Clinic in Berlin galvano-diagnostically, has reached the conclusion that in 80 to 90 per cent. of such children galvanic hyperirritability is present in the Thiernich-Mann sense. Among the 167 children examined there were many in whom spasm of the glottis was recognized by a simple "sighing" or "sucking" during crying or excitement.

This relation between laryngospasm and the Thiernich-Mann reaction, however, loses in importance by the simultaneous report of Japha that 53 cases among 304 children examined, who were free from laryngospasm and mechanical hyperirritability of the nervous system, some of them over three years of age, revealed an increased galvanic irritability in the Thiernich-Mann sense. These children were always nervous or otherwise sickly. Perfectly normal children never showed this irritability.

These reports of Japha coincide with my observations in my article on the Myotonia of Nurslings regarding the findings in older children. In them also, although not employing the new method which was used by Thiernich and



Japha, I repeatedly demonstrated a galvanic hyperirritability of the peripheral nerves (CaCC below 1 Ma. in the facial nerve) in the absence of any other symptom of tetany than the facial phenomenon, and to this I do not by any means attach the importance of a positive symptom of latent tetany. Finkelstein also, as we shall hear later, has very frequently found the Thiemich-Mann reaction in older artificially-nourished infants who did not present the slightest indication of any other hypersensitive condition.

Instead of concluding from this that there are children *free of tetany* who show the Thiemich-Mann reaction, and of placing this kind of galvanic hyperirritability in early childhood upon a different plane from that of the tetany of adults, children without other tetanic symptoms who presented the Thiemich-Mann reaction were declared to be the subjects of tetany and were supposed to possess a tendency to laryngospasm. The important fact in this is that a number of cases examined by Japha who showed the Thiemich-Mann galvanic hyperirritability without other symptoms of tetany were over three years of age. At this age, according to my experience at least, the tendency to spasm of the glottis has disappeared. Children who have not been affected by that condition before the end of the first year of life are not subject to it later.

Before investigating the individual symptoms which are associated with tetany, in regard to their actual relation to this spasmodic neurosis, a positive standpoint must be established, which culminates in the fact that there is no separate division for the tetany of infancy, and that even in childhood we may only refer to this affection when the above-mentioned symptom triad is present: *spontaneous or artificially-produced spasm of the extremities with distinctly demonstrable galvanic and mechanical hyperirritability of the motor nerves*. In my opinion the difference between manifest and latent tetany, even in infancy, is that in the former spontaneous spasms of the extremities occur, and in the latter only spasms artificially produced. Latent tetany, however, may at any moment become the manifest form of the disease.

Here, as was previously indicated, we are opposed to most modern authors, among whom, however, there is no unanimity in regard to the conception of latent tetany.

Ganghofner has divided the symptoms of infantile tetany which are present in the interval free from attacks into *obligatory* and *facultative* latent phenomena, and to an especially increased mechanical irritability and to Trousseau's phenomenon he has attached the importance of obligatory latent symptoms. Spasm of the glottis and eclamptic conditions he has declared to be facultative latent phenomena of *tetania infantilis*.

Thiemich, on the other hand, attaches principal importance to the galvanic contractility which results from the cathodal opening contraction (CaOC below 5 Ma.).

Escherich, again, has regarded all forms of spasm occurring in rachitis which run their course with general hyperirritability as the "tetany of rachitis."

I maintain that the diagnosis of tetany in infancy cannot be considered

from other viewpoints than those which predominate at other periods of life. The only positive criterion of latent tetany is the possibility of artificial provocation of a spasmodic condition analogous to a tetanic seizure. Mechanical hyperirritability of the nerve trunks occurs in infancy without Trousseau's convulsion, as we have already seen, and this is also true of those children who possess an increased galvanic irritability.

These circumstances are not altered by the fact that the tetany of children in its etiologic aspect must be regarded from another standpoint than that of adults, because, like all other forms of spasm dependent upon hyperirritability, in early infancy it is in intimate relation to a disease which is foreign to advanced age, namely, *rachitis*.

Therefore, while it is certain that in a great number of cases of laryngospasm the facial phenomenon and Erb's or the Thiernich-Mann symptom is present, the view is by no means justified that all cases of spasm of the glottis are tetany; on the contrary, it only demonstrates that numerous rachitics suffer from symptoms of a hyperirritable nervous sphere.

It has been several times reiterated in regard to the facial phenomenon that undoubtedly quite a number of children who suffer from laryngospastic attacks do not present this sign and it is necessary to add that the facial phenomenon no longer is of decisive importance in the diagnosis of tetany. During this period of life it is found in hysteria, in night-terrors, and also in children who are merely anemic. It is a symptom of a hyperirritable nervous sphere and is not necessarily associated with tetany. Kraepelin and I were able to demonstrate Chvostek's phenomenon in myxedema, and I found it in mongolism. Loos, H. Schlesinger and Escherich noted it in children free from tetany but otherwise sickly. Frankl-Hochwart saw it in 4 per cent. of normal children.

There are severe cases of rickets in which repeated attacks of apnea have occurred but never the facial phenomenon nor an increase of galvanic irritability in Thiernich's sense. Children of this kind show a conspicuous tetanic rigidity over the entire body during the attacks of apnea, wherein the hands present a spasmodic closure of the fingers. Nevertheless in the intervals free from attack these children do not show the characteristic contraction of the hand of tetany inasmuch as Trousseau's symptom cannot be produced artificially.

In my opinion galvanic hyperirritability in infancy, and particularly the Thiernich-Mann reaction of the peripheral nerves, is of somewhat different importance than in later life. Above all, it is noted with great frequency in psychically hypersensitive rachitic children who suffer from the convulsions previously designated as idiopathic; further, as was shown by the investigations of Finkelstein and Japha, in nurslings who are otherwise normal under the influence of certain deleterious alimentary effects which arise from *nutrition with cow's milk*; thirdly, as was found by Japha and myself, galvanic hyperirritability is present in children who, beyond the age of rachitis, are free from rickets, free from hyperirritability convulsions and also free of those

alimentary difficulties which are supposed to be due to nourishment with cow's milk in infancy. Under such circumstances it is necessary to seriously consider whether it is permissible to regard this as an obligatory symptom of latent tetany in infancy, or whether, on the contrary, it belongs to the special peculiarities of early infancy, showing its effect in various general disturbances by a hyperirritability of the central nervous system which can be demonstrated in a measurable manner by the reaction of the motor nerves to the galvanic current.

Here, in my opinion, the conditions are different from tetany of adults. I do not know whether a modern neurologist would designate a person presenting galvanic hyperirritability, without other symptoms of tetany, especially without Trousseau's sign, as a subject of latent tetany; but this much is certain, if an adult has manifest tetany he also presents galvanic hyperirritability. If the history of tetany is investigated it will be found that the mechanical hyperirritability of the peripheral nerves was the particular reason for Erb's attempt by galvanic methods to bring this hyperirritability into a definite numerical relation. If it is true that a great number of children in the first years of life, under various conditions—in consequence of rickets, of improper nutrition, of other debilitating influences upon the nervous system—present this galvanic hyperirritability which in later life is found only in tetany, a knowledge of this fact is necessary, but all of these children need not be designated as candidates for tetany or as the subjects of latent tetany. On the contrary, galvanic hyperirritability is to be regarded only as a part phenomenon of that general hyperirritability of the nervous system of infants which is due in the first years particularly to the rapid growth of the brain.

In my opinion the same sharp limits should be extended to the conception of tetany in infancy which obtain in the special pathology for the later periods of life; without Trousseau's symptom or carpopedal spasms no diagnosis of tetany; but we must again insist that mechanical and galvanic hyperirritability of the motor nerves is never absent in infantile tetany.

The conceptions and terms "spasm of the glottis" and "eclampsia" of infancy must be retained as substantive designations for definite forms of convulsion, just as the conception of tetany, in which, however, they are not to be included. In addition we must always bear in mind that these three varieties of spasm may occur simultaneously in the same individual.

Mechanical and galvanic hyperirritability is always present in tetany, *sensu strictiori*, but is inconstant although very common in simple spasm of the glottis and in eclamptic convulsions—briefly, in all of those affections which we have learned to recognize as hypersensitive conditions of the first years of infancy.

The fact that the definition of tetany is viewed in various ways by different authors is a great drawback in determining the *frequency* and the occurrence of tetany in infancy and in the utilization of reports. Anyone, like ourselves, who only regards those cases as tetany in which characteristic tonic spasms exist in the extremities will find that tetany is a relatively rare affection, some-



what more frequent in certain regions, than in others, just as is the tetany of adults. On the other hand, those who include the entire group of hyperirritability convulsions and in addition all children who present galvanic and mechanical hyperirritability of the motor nerves in tetany will find much greater figures. Thus, according to the unanimous reports, genuine tetany is a rare disease in Switzerland and in France, while laryngospasm and eclampsia are no less frequent there than elsewhere.

Great variations in the figures of tetany of two observers in the same city can only arise from a different conception of the disease. Thus, Heubner found only 3 cases of tetany among 17,000 sick children in Berlin; Baginsky, in the same city and during the same time, 143 cases among 25,000 children.

In addition to the designation "*latent tetany*" the term "*tetanoid condition*" has been invoked for a number of cases which present symptoms of hyperirritability of the nervous sphere. Escherich refers to a tetanoid condition in children who show laryngospasm and the facial phenomenon. Thiemich has lately attempted, in a French publication, to differentiate the convulsions occurring in infancy, according to the galvanic reaction, into those with and without the tetanoid condition. This author, however, must admit that there is no difference in the manner in which the spasm presents itself in hypersensitive children with a tetanoid reaction and in those with a normal galvanic irritability.

The expression "tetanoid condition," to designate children with a hyperirritable galvanic or mechanical reaction does not seem fortunately chosen, because it does not cover the conception which the author of this term, v. Frankl-Hochwart, understood by it. v. Frankl-Hochwart designated as a tetanoid condition the occurrence of Trousseau's phenomenon and paresthesias in the hands and fingers in tetanic patients during the interval free from attacks.

If the term "tetanoid" were to be employed in another sense in infancy it would be necessary to refer expressly to a "tetanoid condition of children," which is not synonymous with the tetanoid condition of adults. I believe it is advisable not to extend the conception of the tetanoid condition so far as was done by Escherich and Thiemich, but simply to refer to hyperirritable children—a designation which is certainly not prejudicial.

Genuine tetany of infancy, in regard to its appearance, is largely the same as in adults. There can be no doubt that here also epidemic conditions prevail. There are large cities in which the picture of genuine tetany in nurslings and children is unknown, and Hecker, of Munich, has reported in the Volkmann collection, in regard to the tetany of children in his city, that his conclusions were derived principally from the study of the literature and not from his own observations. The tetany of older children differs neither etiologically nor clinically from that of adults.

No other form of hyperirritability convulsions of young children is so associated with *season* as is genuine tetany. According to my careful investigations it is present in Vienna *in the late winter and spring exclusively*, and here again there is a more cumulative appearance of tetany cases within the desig-

nated months in certain years, as in 1899 and 1900, than during the same months of other years, as in 1902 and 1903 when there was a remarkable decrease in their number.

The other forms of hyperirritability convulsions—laryngospasm and eclamptic attacks—are forms of hyperkinesia of young children which occur everywhere and in every season of the year, although more frequently in the late winter and spring. They therefore possess a certain relationship with tetany without being analogous conditions.

#### D. SPASM OF THE HEAD AND EYES IN CHILDREN: SPASMUS NUTANS AND NYSTAGMUS

These two functional spasms of children must be considered together, as they are always associated in infancy and are also combined genetically. As a rule nodding spasms appear at an earlier age than tremor of the eyes and very often disappear before the latter condition so that spasm of the eyes occasionally is present alone for a limited time.

Both forms of spasm occur as part phenomena of organic affections of the brain, and in adults and older children are also observed in epilepsy (Lange). These spasms, occurring in the first three years of life, are almost exclusively to be regarded as *functional neuroses*, which, like laryngospasm, eclampsia, and tetany, are associated with general *hyperirritability* of the nervous sphere.

*Spasmus nutans* is a form of spasm of purely functional nature peculiar to the first three years of life, which was first described in 1850 by Hensch and Romberg, but awakened greater interest among pediatricists after the minute investigations by Kassowitz, Lange, and Raudnitz.

The following is a brief description of the condition:

The affected children show a peculiar *shaking of the head*, either in a vertical direction, in the form of *nodding* or *bowing movements*, whence the names *nodding* and *salaam spasm*; or the head wobbles from side to side or performs rotary movements in the atlanto-occipital joint. Certain children, while awake, show one or another variety of this spasm continuously, others are affected periodically. According to Raudnitz the shaking or rotary movements are not particularly rapid, at most with the rapidity of the seconds pendulum. Here the lightning-like appearance which is characteristic of the clonic convulsions of the muscles of the extremities in children, together with loss of consciousness, is entirely lacking. In the affection under discussion consciousness is always retained. The shaking movements generally disappear when the child is unobserved and in complete repose. With intended movement or upon attempts to fix certain objects, or under psychological irritation, the spasmodic movements of the head become more severe and as a rule the first excursus at the onset of an attack is more rapid and complete than those which follow.

So far as I have been able to determine the affection does not develop suddenly, but very gradually, the head now and then wobbling to and fro with an erect position of the body. In most cases but little attention is given to the affection at its onset. The first attacks consist of a few swaying motions of the

head, lasting scarcely a minute, which are supposed by those about the patient to be intended or the signs of an especially pleasant or painful stimulation. In time, however, these movements become more severe, prolonged, and recur several times daily, particularly under excitement. Usually importance is not attached to the case until the attacks occur more frequently and are more prolonged.

In the majority of instances the nodding spasm is associated with *tremor of the eyes (nystagmus)*.

This form of spasm consists of *clonic contractions in the course of the ocular muscles* in rapid sequence. The excursions may be so rapid that we might actually speak of tremor of the bulbi. The contraction of the eyes occurs in various directions according to the nature of the predominant contracting muscles. It may be either in the vertical or in the horizontal plane, or occur in both directions alternately. In addition to the permanent tremor there may also be a rapid to-and-fro movement of the bulbi. Severe cases of spasmus nutans are always associated with nystagmus; only in the stage of onset, as a rule, is tremor of the eyes absent. In some cases nystagmus is the chief symptom of the combined convulsive category under consideration and becomes continuous, while nodding and bowing spasms occur in transitory and very brief attacks. In other cases the child is attacked by nystagmus as soon as its attention is fixed, while the shaking of the head appears only when there is simultaneous excitement. Upon close questioning I was able to determine that nodding spasm had previously been present even in those cases of nystagmus which were unaccompanied by the phenomenon at the time of examination. Nystagmus is not always developed equally in both eyes.

Both forms of spasm disappear during sleep. In the waking hours many children show both varieties of spasm uninterruptedly or broken by very short pauses. Often, while recumbent, the child only shows nystagmus, but as soon as the head is raised or the body brought into a vertical position spasmus nutans appears. In all cases the movements of both the head and the eyes are increased by psychic stimulation. These varieties of spasm run a favorable course in infancy provided they are pure functional neuroses. The spasmodic condition disappears during the third or fourth year of life without resulting mental disturbance, paralysis or contractures, provided the *underlying affection, rachitis*, is recognized and *treatment by phosphorus* instituted.

In regard to the development of these spasmodic affections it must be remarked that children with nodding spasm and nystagmus always present other symptoms of a hyperirritable nervous sphere, as do those who suffer from spasm of the glottis and eclampsia.

Furthermore, I have found, in quite a number of cases of spasmus nutans, that the affected child suffers simultaneously from the two last-named conditions.

Among our cases, which include more than 50 children with spasms of the head and eyes, we have never failed to find rachitic changes in the skeleton, so that it appears necessary to include spasmus nutans and nystagmus in the



category of hyperirritability convulsions of infancy associated with rachitis. Here we must not entirely ignore the view of Raudnitz that over-exertion of the muscles of the eye, as the result of *insufficient or bilateral light*, plays a certain rôle in the occurrence of these forms of spasm similar to that in the nystagmus of miners.

It is easy to concede that in hypersensitive children who are at the age of most intense growth of the brain, those ganglion groups and conduction tracts become particularly irritable which are exposed to special exertion.

Accordingly we would then be dealing with a functional hyperexertion, especially an increased irritability of certain cerebral motor nerves (ocular nerves and spinal accessory).

Analogous to the assumption of Kassowitz of a cortical origin of spasm of the glottis, Stamm has demonstrated that in nodding spasm and in nystagmus of children we are concerned with a mechanical irritation of certain portions of the cerebral cortex due to hyperemia of the internal periosteum of the skull resulting from rachitis. In any event the predilection of age between six months and three years, the never-failing rachitis, the constant psychological hypersensitiveness, and the association with other spasms of excessive irritability, make it reasonable to include also the nodding spasms of children in the realm of hyperirritability convulsions.

Supplemental to the foregoing, it must be mentioned that **cataleptic** phenomena have been repeatedly observed in *rachitic* children without organic disease of the central nervous system. To Epstein is due the credit of first remarking these conditions, which I am in a position to confirm. Here we are concerned with markedly rachitic nurslings who present the most grotesque positions of the extremities, which are retained for minutes if the limbs are passively arranged, and are only gradually replaced by a normal attitude. I saw a child of this category, aged nine months, who presented cataleptic rigidity of the extremities in addition to laryngospasm, nodding spasm and nystagmus. In this case a lower limb which was completely extended at the knee-joint and flexed at a right angle at the hip-joint, remained for minutes in this position—an absolutely unnatural one which can be maintained voluntarily only with the greatest difficulty.

#### ETIOLOGY OF HYPERIRRITABILITY SPASMS

In turning to the *etiology* of hyperirritability convulsions of infancy it is first necessary to emphasize that in my opinion *rachitis* is the connecting link for all of the forms of spasm dependent upon general hypersensitiveness of the nervous sphere. This affection has never been absent in any of the cases under consideration. This view, already approached by Elsässer and Gay, and in recent times warmly advocated by Kassowitz and his pupils and also by Escherich, is opposed to a second theory, according to which the spasms here under

discussion, or the hyperirritability which is the foundation of them, are due to *alimentary* damage and are primarily dependent upon *gastro-intestinal auto-intoxication*. A number of authors, particularly Comby and D'Espine, have gone further, in that they regard *gastro-intestinal auto-intoxication* as the common cause of rickets as well as of the hyperirritable conditions of infancy. Others, again, have entirely ignored a relation between rickets and hyperirritability convulsions and have declared the occurrence of the latter with rickets to be an accidental combination, and that the spasms alone are disturbances of the nervous sphere due to alimentary damage. .

In my opinion weighty factors oppose the generalization of auto-intoxication as the etiologic principle of hyperirritable conditions and spasms in infants. We may conceive that, in acute digestive disturbances, with the entrance of the enterogenous poison into the circulation an irritation of the central nervous system arises which shows itself by intermittent clonic spasms. It is also conceivable that in chronic digestive disturbances the toxic substances formed and absorbed in the intestinal canal give rise to intermittent spasmodic conditions as often as an intense over-flooding of the organs with toxic products arises. Again, it is possible that under certain conditions persistent tonic, not intermittent spasmodic, conditions may occur from the continued absorption of intestinal poisons into the circulation in chronic auto-intoxication.

The best example of this form of autotoxic-spasmogenic action is the previously described myotonia perstans of nurslings in severe intestinal affections. The autotoxic spasmodic conditions just mentioned are controlled by the presence of a demonstrable and recognizable intestinal disturbance leading to auto-intoxication, and therefore exist for a relatively limited time, disappearing when no more toxic products are produced from the gastrointestinal tract, as has already been carefully explained in the discussion of occasional convulsions of childhood.

It is rather remarkable that, in those children particularly who suffer from authentic spasmodic conditions of gastro-intestinal origin dependent upon endogenous toxin formation within the intestinal canal, a permanent hyperirritability of the nervous sphere in the sense of our previous explanation is only demonstrable when the children were over-sensitive from birth, and, naturally, are also occasionally affected by the characteristic spasms of an intestinal affection.

Children of the last category for many months, occasionally for years, suffer from a hypersensitive nervous sphere which is characterized by permanent demonstrable symptoms and especially by intermittent respiratory spasms.

If the hyperirritability which is the basis of these spasms is made dependent upon gastro-intestinal auto-intoxication, then the spasms in these cases must last for months, and could only be due to chronic diseases of the chylipoietic system or from a continued deficiency of the intermediary metabolism. But it must be stated emphatically that disturbances of the last-mentioned nature are demonstrable only in a very small fractional proportion of hyperirritable

infants, while, inversely, hyperirritable conditions, especially laryngospasm and eclampsia, also occur in healthy breast-fed children who do not suffer from intestinal disease, and those who are decidedly atrophic almost never show such hyperirritable and spasmodic conditions. Further, if breast-fed children suffer from a general hyperirritability of the nervous system this certainly does not occur during the time in which the appearance of intestinal intoxication could be most readily explained—during the period of weaning; on the contrary, its first appearance is much earlier, about the fifth or sixth month, and, as has been already explained, in the form of characteristic *respiratory spasm in psychical irritation*. Such children, however, are *never free from rickets*. The reason why hyperirritable conditions are almost never observed in atrophic children is based upon their lesser predisposition to rachitis.

Children who present the hyperirritable and spasmodic conditions under discussion are usually well-nourished and have a considerable amount of adipose tissue. Aside from rickets they usually deviate from the normal only by a moderate pallor of the skin and slight puffiness of the face. But I have observed these pathologic conditions also in splendidly nourished children of good color, although always in breast-fed children suffering from rachitis.

On the basis of electric investigation of the motor nerves Thiemich, Gregor and Finkelstein attempted to refer the hypersensitiveness of nurslings, and with this also the so-called infantile spasms, to the *nutrition*.

Thiemich emphasized that the *galvanic irritability* of the motor nerves in one and the same child is subject to great modifications, as Gregor has shown, under the influence of nourishment. Thiemich also pointed out that, in artificially-nourished children who suffer from constipation, by emptying the intestinal tract or by the institution of a simple water diet the galvanic hyperirritability previously present may be decidedly decreased, and that the change to natural food prevents its recurrence. From this may be deduced important conclusions for treatment, which primarily concern the intestinal canal as the point of attack. Finkelstein has reached similar results and has attached to artificial nutrition with *cow's milk* a predominant importance in the occurrence of the tetanoid condition. Thus, upon the basis of electric investigation, the old views promulgated by Flesch and Rehn, and recently again made prominent by Fischbein, were revived, which required as curative measures for spasm of the glottis and eclampsia in artificially-nourished children a change of food and a return to breast milk. Finkelstein, in particular, believed that from the *whey* of cow's milk the elements responsible for the hyperirritability reached the circulation of the nursling, to which Thiemich responded that he was able to demonstrate galvanic hyperirritability in children who had never taken a drop of cow's milk, but in addition to breast nourishment had been fed with starchy substances.

Although I am not in a position to confirm the connection between Thiemich's tetanoid condition and galvanic hyperirritability and artificial nutrition, as I have not investigated these relations, nevertheless, the fact is certain that eclampsia and laryngospasm are very common in breast-fed children and



are much less frequent in those who are artificially nourished. If nourishment with cow's milk, or the addition of a few biscuits and some flour soup to breast milk were capable of producing a tetanoid condition in an infant it would be difficult to explain why so many artificially-nourished children are permanently free of hyperirritability convulsions.

In view of these circumstances the question is justified, whether the presence of galvanic hyperirritability in the Thiemich-Mann sense may be identified only in early childhood with the "tetanoid condition" or with "latent tetany."

If it is true that nutrition with cow's milk and other alimentary deleterious agents which provoke the reaction of the peripheral nerves in the nursing, produce a tetanoid condition, there would be a disagreement between the time of appearance of the hyperirritability convulsions and of galvanic hyperirritability. The alimentary damage in artificial nutrition would have to be principally operative in the warm seasons. Accordingly, galvanically hyperirritable children should be found most frequently in mid-summer and in the autumn. At this season of the year, however, the so-called idiopathic convulsions of infancy are least frequent, while children are most commonly attacked by tetany, laryngospasm, and eclampsia in the late winter and spring. From this it appears that too far-reaching conclusions should not be constructed in regard to a connection of galvanic hyperirritability in early childhood with idiopathic infantile convulsions.

There is another factor in opposition to the supposed spasmodic effect of cow's milk. Psychical hypersensitiveness of children usually disappears at a time during which cow's milk is the principal nutriment—in the latter half of the second year, or at least much earlier than the beginning of a diet deficient in cow's milk, similar to the food of an adult. These conditions disappear, rapidly and independently of nutrition, under the influence of *treatment with phosphorus*, or when the rachitis improves spontaneously. This experience, confirmed in thousands of cases in our Clinic in Vienna, is alone sufficient to deprive the alimentary theory of hyperirritability spasms of infancy of all support. Anyone familiar with the conditions under which children of the lower classes develop and who must concern himself with such children professionally, knows that advice to patients of that kind in regard to a change of food is of exceedingly problematic value. If, notwithstanding this, the administration of phosphorus has a constant and occasionally even miraculous effect in these hyperirritability convulsions, the excessive excitability which is the cause of them must be due to other circumstances than to the character of the nourishment of these infants.

In this entire controversy one factor deserves emphasis, and perhaps is the most important in the etiology of infantile convulsions. Infantile convulsions are relatively rare among the better classes and I have never observed true manifest tetany and spasmus nutans in children of my private practice. In hospital practice, however, eclamptic conditions and spasm of the glottis are of daily occurrence, immaterial whether the patient is fed artificially or at the

breast. Whenever I have observed laryngospasm and eclampsia in well-nourished breast-fed children of the well-to-do they were always rachitic and were kept for a long time uninterruptedly in close rooms, as is common in prolonged cold weather among indulged city children. Therefore, in the better class of private patients particularly, hyperirritable conditions appear in nurslings in the late winter and spring simultaneously with the development of *rachitis of the skull*. In reviewing all of these experiences I can recognize an exciting cause in the alimentary damages which affect the nursling for the individual spasmodic attack, but not the basis upon which the pathologic condition of general hyperirritability develops. In my opinion the fact is not altered that galvanic irritability of the peripheral nerves among infants nourished with cow's milk is more decided than with natural food.

The entire theory of the connection of hyperirritability convulsions of children with nutrition stands upon an insecure foundation. Naturally, it cannot be denied that psychically hypersensitive children are attacked by eclamptic conditions during acute and chronic digestive disturbances, as, in patients of this kind particularly, any derangement of health is calculated to originate convulsions. I am firmly convinced that children who, in their earliest youth, have suffered from hypersensitiveness, retain a certain tendency to convulsions for years, and that these children are liable to spasmodic attacks in any disease, immaterial whether it has to do with nutrition. I have observed the facial phenomenon in three cases from the age of infancy to beyond the fifth year of life in children who were previously eclamptic; after the third year the laryngospastic attacks no longer occurred, but a convulsive seizure appeared upon the occasion of any febrile disease.

Therefore, we are of the opinion that rachitis alone, that is, the deleterious agencies which give rise to it, is the basis of the hypersensitiveness in question. I am fully justified in maintaining that *without rachitis there are no hyperirritability convulsions of children*. This pertains to the simple forms of this convulsive category—spasm of the glottis and eclampsia—as well as to tetany and spasmus nutans. Most authors, even those who deny the theory of Kassowitz, must admit that in an overwhelming percentage of cases the children affected by spasm are the subjects of rickets. Escherich, whose pupil Loos formerly denied any connection between spasm and rachitis, has characterized the spasmodic condition of nurslings dependent upon hypersensitiveness of the nervous system as "*tetany of the rachitic*." In fact the only question to be decided is whether rickets as such causes hyperirritability in these children, as Kassowitz believes, or whether the same deleterious agents which give rise to rickets are also the cause of general hyperirritability. In any event the periodic coincidence of the frequency curve of rickets and of hyperirritability convulsions, upon which Kassowitz has laid particular stress, proves an intimate correlation of these two pathologic conditions.

In this respect the fact that various authors show different percentages is due principally to the lack of uniformity in the valuation of rachitic symptoms. Above all, many authors make the mistake of deferring a diagnosis of rickets



until the child shows a decided rosary and a curvature of the bones of the extremities, therefore when the rachitic phenomena are so obvious that we are forced to recognize them. It is also a common belief that rickets of the skull should only be assumed when marked *craniotabes* exists. Therefore minor grades of rickets or cases of rickets of the skull in process of retrogression are overlooked. Naturally it would lead us too far to enter in detail upon the symptomatology of the rachitic osseous changes, but it must be remarked that *soft sutures of the cranial bones and protuberances of the costal cartilages* are sufficient criteria for a diagnosis of rickets.<sup>1</sup> These are the first changes in the rachitic skeleton which can be demonstrated by palpation. If we assume, with Kassowitz, that the rachitic bony changes are due principally to the influence of atmospheric conditions, such as obtain in foul respiratory air (the so-called odor of the poor), and that the same respiratory deleterious factors which give rise to rickets are also responsible for nervous hypersensitiveness, the fact is explained that in a certain number of cases the first noticeable spasmodic conditions appear simultaneously with the first palpable signs of rachitis.

In regard to the relation of *craniotabes* to spasm of the glottis, the gross mechanical explanation of Elsässer that external pressure upon the surface of the brain in a yielding skull is a factor in the production of spasm has in my opinion been quite improperly rejected, for, as Heubner has lately remarked, in exceptional cases such a possibility must be considered.

The principal connection between *craniotabes* and hyperirritability convulsions is that *craniotabes* is an *early symptom of rickets of the skull*.

Most opponents of the theory of an association of the so-called idiopathic convulsions of young children with rickets of the skull have claimed that there are many cases of *craniotabes* in which spasmodic conditions are never observed, and that, inversely, many rachitics who do not present *craniotabes* develop eclamptic-laryngospastic attacks. Comby asserts that among more than 50 cases of *craniotabes* which he collected in the year 1893 only one case of spasm of the glottis and two cases of eclampsia were observed. In view of our previous explanation these figures of Comby's are not convincing; the only point of importance is whether the spasms in question appeared in rachitic subjects or in children who were free of the malady.

In my opinion a certain parallelism exists between the sensitiveness of the osseous and nervous systems during their period of energetic growth and development, in the second year of life. As cranial rickets, which is characterized by an especially intense growth of the skull, shows a particular tendency to develop at this period because, during the active processes of growth, the points of opposition of the bony system appear to be unusually sensitive to injuries of a general nature, we may also assume that the brain substance, the growth and development of which approximates the development of the skull, is likewise vulnerable during the same time.

Perhaps a closer parallelism exists between the rachitic bony disturbances

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<sup>1</sup>See article on "Rickets," this volume.



and the hyperirritability of the nervous system. It might be assumed that the same deleterious factors of general nature which incite the rachitic tissue-irritation in the intensely growing bones at their points of apposition produce a change in the developing brain which reveals itself by hypersensitiveness. Accordingly the hyperirritability may be regarded as *the effect of the rachitic damage of the infantile nervous system*.

In the general discussion of these affections the theory of Kassowitz has been mentioned, which refers the spasmodic condition and the psychical hyperirritability of rachitic children to a cortical hyperemia due to cranial rickets. Escherich believes the *respiratory damage* which Kassowitz has regarded as important in the development of rachitis to be also influential in the etiology of the convulsive conditions of children and that the coincident appearance of the curve of rickets and of spasm in the late winter and spring is to be referred to the prolonged effect of the same noxa, namely, of respiratory influences in the insufficiently ventilated and thickly populated quarters of the lower classes during the winter.

Concerning the relation of idiopathic convulsions to rachitis it is immaterial whether Kassowitz's theory of meningeal and cortical hyperemia is accepted as the cause of spasm in the rachitic child. As Kassowitz regards the rachitic changes of the bones as a hyperemic inflammatory process which is particularly prominent in early childhood, especially in the bones of the skull, he concluded, on account of the intimate connection of the dura mater with the cranial bones, upon a propagation of an inflammatory hyperemia of the latter to the meninges and the cortex of the brain. The entire series of symptoms of psychical hypersensitiveness of rachitic children—sleeplessness, fright, restlessness, sweating of the head, shaking spasms, contractures, and particularly spasm of the glottis—is the expression of an abnormal irritability of the central nervous system dependent upon a cortical hyperemia. Kassowitz based his conclusions upon the experimental investigations of Semon, Horsley and Krause, who, by faradic irritation, found a center in the cerebral cortex for the closure of the glottis, and of Unverricht and Preobraschensky, who discovered a cortical center for the expiratory arrest of respiration.

In opposition to Kassowitz's theory that *cortical* irritation produces respiratory spasms in rachitics, the assertion has been made that the hyperemic condition of the cerebral cortex and of the meninges at the convexity, due to rickets of the skull, must be general when it exists at all, and that therefore the special preference for a hyperirritation of the cortical centers of the respiratory muscles is not clearly understood. In my opinion, however, this relation is quite comprehensible.

If, as has been indicated, a general hyperirritability of the cerebral cortex must be assumed as the foundation of all convulsive conditions under consideration, for the stimulation of an attack, as a rule, certain causative factors which quickly irritate the nervous system of the child are essential, such as fright, anger, sudden cooling, pain, and the like. I need only call attention to the effect of similar irritating factors upon the nervous system of adults. Here

also tonic spasmodic conditions in the muscles of the larynx and of respiration play a special rôle. Loss of speech from fright, hindrance of respiration in extreme cold, and the like, are evidences that in general hyperirritability the centers which preside over the muscles of the glottis and of respiration are much more readily excited than any of the others. Accordingly there is no factor in the prevalence of respiratory spasms in hyperirritable rachitics which would oppose the view of a general cortical hyperirritability produced by hyperemia of the surface of the brain.

If we enter further into the domain of convulsions we will find that in the hyperirritability spasms no voluntary muscle of the rachitic nursling is exempt from spasm. In the eclamptic attacks occasionally not a single voluntary muscle remains unaffected. Respiratory spasm is present alone only in very mild cases, but tonic carpopedal convulsions frequently begin with a crowing, spasmodic, inspiratory murmur due to closure of the glottis, and it is quite clear that not only the centers for constriction of the glottis and of the respiratory muscles but also the cortical centers of the entire voluntary musculature are in such a condition of hyperirritability that the slightest external cause may be the signal for a convulsion. If loss of consciousness is included in the calculation it is obvious that *the entire cerebral cortex is in a hyperirritable state*, which, in our opinion, is the actual foundation for the disturbance in function of the nervous system of rachitics here under consideration.

We have still to consider a view especially propounded by Escherich, according to which the constitutional disturbance designated as *status lymphaticus* or *status thymicus* is the ultimate cause of the hypersensitiveness of infancy, therefore of the group of convulsions under discussion. According to Paltauf and Escherich the *status lymphaticus* is a general disturbance characterized by a pale and puffy appearance of the skin and by enlargement of the entire lymphatic tissue including the thymus, the spleen, the glands of the tongue, of the pharynx and of the tonsils. In regard to the *thymus* it has been assumed that through a counteraction of the internal secretions of this gland and of the secretions prepared by the thyroid gland an antitoxic effect of the organism is produced, and that a disturbance of this function associated with enlargement of the organ causes a permanent hyperirritability of the nervous system.

The status thymicus has been held responsible for many sudden deaths in infancy, whether justly so is doubtful. Quite as questionable is the relation of the status lymphaticus to hyperirritability of the infantile nervous sphere. Probably the most serious contradiction of this theory is the occurrence of laryngospasm in normal infants in whom no trace of a hyperplasia of the lymphatic tissue can be discovered.

The thymus gland, however, is of importance in the consideration of respiratory spasm in children, as a definite condition of *difficult respiration* may arise from the pressure of the enlarged gland upon the *trachea*. *Stridor congenitus* may be the exclusive consequence of *hyperplasia of the thymus*, as can be determined by X-ray examination, and another respiratory anomaly, *asthma*

*thymicum*, consisting of dyspnea and cyanosis with noisy respiration, is the most intense form of pressure of the thymus gland upon the trachea. But in these processes which run their course with demonstrable enlargement of the thymus the hyperirritability convulsions are absent, especially the typical spasm of the glottis.

It has now been definitely decided that the hyperirritability convulsions of children bear no relation to the *thyroid gland*. The investigations of Sandström, Gley, Vassale, and Generali and Biedl have shown that the *tetany* produced experimentally in animals by extirpation of the thyroid gland is not the result of removal of that body but of removal of the *parathyroid glands*—the so-called *epithelial bodies*; extirpation of the thyroid gland gives rise to *myxedema*, without tetany.

Furthermore, only a small group of hyperirritability convulsions would come under consideration here, namely, the genuine tetany of infancy. At the present time, however, the relation of the epithelial bodies to human tetany is merely an interesting hypothesis which it is well to know, without deducing far-reaching conclusions therefrom.

In several myxedematous and mongoloid infants I was able for a considerable time to demonstrate Chvostek's facial phenomenon, and in a myxedematous child aged 5 months I found tonic carpopedal spasms of tetanic nature which disappeared after treatment with thyroid gland.

### III. DIAGNOSIS

Further explanation regarding the *diagnosis* of the individual forms of functional spasm in infancy appears superfluous, as sufficient space was devoted to it in the clinical description. Only the relation between functional infantile spasms and *epilepsy*, the differential diagnosis between these two varieties of convulsion, and the differentiation between infantile spasms of organic and of functional nature will be considered.

First it is necessary to investigate in detail the relation between infantile *eclampsia* and *epilepsy*, especially as the question arises throughout the entire literature of epilepsy whether there be any connection between this neurosis and the functional spasms of childhood.

In the older literature infantile eclampsia, which to-day is included among the hyperirritability convulsions, is identified with epilepsy, and Baumés, in his monograph of 1805 upon Convulsions of Children, asserted that merely an external difference of course and age existed between infantile eclampsia and epilepsy.

In recent times Féré in particular emphasized the identity of the two forms of convulsion, and remarked that in his own experience epileptics frequently suffered from convulsions in infancy. He found that 34 per cent. of his epileptic patients had had eclampsia infantum. In the reports of various authors the relative percentage of infantile eclampsia and epilepsy differs essentially.



According to Moreau the proportion is 17 per cent.; Haabermas and Berger report 20 per cent. Gowers states that many cases of epilepsy begin in the first three years of life, and estimates such an occurrence in about 12 per cent. of his patients, which does not include simple infantile eclampsia. According to Osler the onset of epilepsy occurs in the first three years in about one-third of all cases.

On the other hand some authors claim to have demonstrated that children who suffered from eclamptic attacks in early infancy afterward became epileptics. Coutts, for example, noted a transition from eclampsia to epilepsy in 7.5 per cent. of eclamptic children for a long time under his observation.

Dufour believes that, of 66 eclamptic children, 15, therefore about one-fourth, subsequently suffered from true epilepsy. It has also been frequently remarked that eclamptic children, even though not epileptic, are afterwards nervous and suffer in later life from migraine, chorea, and other functional neuroses.

Henoch and Heubner were able in several instances to follow the gradual transition of infantile eclampsia into true epilepsy.

Among 54 closely studied cases of infantile eclampsia D'Espine found only 4, therefore 7.4 per cent., of later genuine epilepsy. In these cases the apparently idiopathic eclampsia passed quite unnoticed into the epileptic condition. In one case of D'Espine's in which the convulsions developed in the third year and were supposed to be dependent upon phimosis, the attacks disappeared transitorily after a circumcision in the fourth year, to recur soon afterward with the clinical picture of genuine epileptic convulsions.

An opposite standpoint is taken by Binswanger, in his recent work upon epilepsy, in regard to the connection of that condition with eclampsia infantum. This author admits a hereditary neuropathic influence which is manifest in infancy, but believes the fundamental difference between eclampsia infantum and epilepsy to be that in genuine epilepsy a specific anomaly, known as an epileptic alteration of the central nervous system, must be assumed which consists in a permanent disturbance of equilibrium between the inhibitive and excitomotor centers of the cerebral cortex. On the other hand, Pierre Marie believes that the individual is not born an epileptic, but becomes so. It is assumed that the greatest number of the infantile convulsions which were noted in the histories of 75 to 80 per cent. of his epileptics were due to mild encephalopathies resulting from infectious diseases, which left cicatrices in the meninges or in the cerebral substance that at a later period were a provocation for epilepsy. The latter part of this view, however, cannot receive general recognition. Above all, it is opposed by the fact that the attacks of infantile eclampsia belonging to the category of hyperirritability convulsions by no means depend upon infectious diseases; on the contrary, after existing for some time they disappear without leaving any essential disturbance of the cerebral function.

Further opposed to Pierre Marie's view is the fact that those encephalopathies in particular which are actually dependent upon acquired inflammatory processes of the cerebral cortex in early infancy, for example, Little's disease,

are associated in only a small proportion of cases with epileptic convulsions, while permanent hypersensitiveness of nurslings which is the foundation of genuine eclampsia represents a pure functional disturbance of the central nervous system without an anatomical foundation.

In my opinion there is no *genetic relation* between idiopathic eclampsia of infancy and epilepsy of later life. With the great frequency of infantile convulsions it is not remarkable that a certain proportion of these children are subsequently attacked by epilepsy without any connection between the two conditions being necessary.

Furthermore, if spasmodic attacks during infancy are admitted in the history of epileptics they must be of varying significance. Thus, the occasional convulsions of early childhood have absolutely no value in the investigation of this question because these early convulsions depend merely upon transitory and accidental disturbances of health and do not owe their origin to a general functional anomaly of the nervous system. Accordingly, merely the relation of hyperirritability convulsions of infancy to epilepsy requires discussion.

We are to-day in a position to differentiate between hyperirritability convulsions and epilepsy of early childhood by a consideration of several clinical factors. The presence of spasm of the glottis and apnea, the existence of mechanical and galvanic hyperirritability in manifest rachitis, should all of these factors be simultaneously present, differentiate hyperirritability convulsions of children from epilepsy. In the differential diagnosis, therefore, only those cases of infantile convulsions come under consideration which run their course without laryngeal spasms.

As a matter of fact I believe that convulsions which occur in early infancy without determinate cause and without the characteristic implication of the respiratory muscles which appears in hyperirritability spasms, are to be separated from simple hyperirritability convulsions and regarded as actual infantile epilepsy.

Also we must not build too strongly upon the differentio-diagnostic importance of rachitic spasmophilia with its symptom-complex, as a combination of genuine epilepsy with rachitic hyperirritability is quite tenable. Therefore, there will always be a small proportion of cases of infantile eclampsia in which the possibility of genuine epilepsy cannot be entirely excluded.

If epilepsy subsequently occurs in a patient in whom convulsions existed in early childhood two conditions are possible: either the infantile convulsions were genuine epileptic paroxysms or the individual in question suffered in infancy, as is so frequently the case, from occasional or hyperirritability convulsions which bear no relation to the later epilepsy.

Of the first possibility it must be noted that the repetition after long intervals of simple convulsions, with loss of consciousness and without respiratory spasms or any other demonstrable acute disease, and continued after the period of the infantile spasmophilia, favors the diagnosis of epilepsy. Upon the basis of these considerations I have repeatedly made a differential diagnosis between epilepsy and eclampsia in early childhood.

Occasionally the period of appearance of the first spasm may furnish support for an early diagnosis of genuine epilepsy. Those cases require especial study in which the convulsive attacks appear toward the end of the second year of life, for then there is always a possibility that we are dealing with the first attacks of an actual epileptic affection.

In the question of the connection of infantile spasm with epilepsy it is necessary to consider that severe eclamptic attacks, especially when associated with expiratory apnea, may lead to capillary hemorrhages in the central nervous organ and in the meninges, and that the residues of such hemorrhages may in later life be the starting-point for epileptic disturbances. This may be the explanation of the transition of apparently genuine eclampsia of infants into true epilepsy, which has been repeatedly reported by trustworthy authors.

It cannot always be determined positively whether the epileptic attacks of early childhood depend upon actual, anatomical, cerebral affections or merely upon a functional hypersensitiveness of the centers. To the weak resistance of the nervous system in early infancy is due the association of very slight anatomical lesions with spasmodic conditions, which disappear as the development of the nervous system advances, but may recur years later under circumstances which cannot be readily ascertained, and particularly at a period when greater effort is required in the mental activity of the child. This accounts for the interval, often of years, between epileptoid infantile eclampsia and later epilepsy, although the period of quiescence is frequently only an apparent one. As a rule, however, such children possess an especially exaggerated nervous irritability, with a tendency to severe outbreaks of anger, to hallucinations, and to night terrors.

The literature contains many statements that *organic affections of the brain* may lead to convulsive attacks which show great similarity to functional infantile spasms.

In my opinion this can only refer to occasional convulsions. In regard to hyperirritability convulsions, apart from nodding spasms there is no possibility of confusion.

In support of this statement, in convulsions due to organic conditions laryngospasm and apnea are almost always absent. Further, in organic cerebral affections of children the clonic spasms of the extremities, as a rule, do not show the uniform distribution over the entire musculature as in functional infantile spasms. It may be difficult to decide whether convulsive attacks which appear during a febrile disease are to be regarded as occasional febrile convulsions or as due to an organic affection of the brain. For example, a child suffering from pneumonia may be attacked by convulsions either of simple, toxic, febrile nature or due to a complicating meningitis. There are no general laws for the solution of such differentio-diagnostic perplexities. Here the differentiation of the convulsions must be considered in connection with that of the underlying affection. In nurslings with an open frontal fontanelle great tension and prominence in that area in the interval free from attacks favors an organic intracranial affection.



The question of organic or functional composition in some cases of *spasmus nutans* associated with nystagmus may cause considerable difficulty, as this condition has repeatedly been observed in chronic inflammatory affections of the brain or cerebral tumor, and also in epilepsy. As a rule it may be assumed that nodding spasms which continue beyond the fourth year of life are associated with organic changes in the brain. The *spasmus nutans* which appears later frequently depends upon hysteria, but then runs its course *without nystagmus*.

If spasms appear in encephalitic cerebral affections or in tumor of the brain, focal symptoms are usually present in the free intervals which exclude a purely functional nature of the spasm. Here also laryngospasm is exceedingly rare except in the case of a combination of rachitic spasmophilia with an organic cerebral affection.

#### IV. PROGNOSIS

In the *occasional spasms* the prognosis as regards recovery depends altogether upon the severity and the gravity of the underlying affection. The spasmodic attack alone only exceptionally leads to a fatal issue and in my experience has occurred only in one affection, whooping-cough. Here, however, as already mentioned, we must always reckon upon the possibility of an effusion of blood into the brain, which may arise in combination with spasms during severe attacks of pertussis. Nor can it be said that the onset of convulsions during infectious diseases indicates a particularly serious course, since children with very mild febrile disturbances are seized with convulsions, and after the affection has run its course are restored to complete health.

With the second group of occasional spasms, which we have learned to recognize as *myotonia* of infants, the condition is somewhat different. Here the permanent spasms of the muscles of the extremity are usually of unfavorable prognostic import as regards the course of the underlying affection, since they indicate a severe toxic condition of the organism. To this statement an exception is made in hereditary syphilis, in which myotonic spasms are observed even in mild cases, which are relieved, simultaneously with the underlying affection, by antisiphilitic treatment.

The prognosis of *hyperirritability spasms* depends upon three circumstances:

1. The importance of the individual spasm;
2. The effect of the same upon the general hyperirritability of the nervous sphere;
3. The connection of the latter with the neuroses of later childhood.

Concerning the first point attention has already been directed to the gravity of the various hyperirritability convulsions, especially the respiratory spasms with the type of *expiratory apnea*. Here, in contrast to the occasional convulsions, the spasm itself is the ominous factor, whereas the underlying disease, rachitis, is by no means associated with danger to life.

In regard to the prognostic importance of the individual spasmodic conditions the rule may serve that those cases are to be regarded as favorable in which only inspiratory laryngeal spasms occur, while children in whom the expiratory arrest or cessation of respiration appears often are always in a certain degree of danger.

As regards the importance of hyperirritability convulsions of children in general the prognosis is chiefly dependent upon the involvement of the respiratory muscles during the seizures, and as the convulsions are more intense in the early life of the child than after the second year, the danger in the first two years is correspondingly greater. Also those forms of spasm in which loss of consciousness plays an essential part are of more unfavorable prognostic import than those in which the irritation is principally of subcortical nature. Accordingly the genuine tetany and nodding spasms of children warrant a favorable prognosis.

In the introduction to this article it was explained that the convulsive children of the first years of life very commonly are the nervous and hysterical subjects of later childhood. It appears that the hypersensitiveness of the infantile nervous system which arises early is not altogether proportionate with the arrest of the convulsions dependent upon it and with the disappearance of the mechanical and galvanic hyperirritability which may be present.

Although, as has been stated, I cannot assume a definite connection between epilepsy and hyperirritability spasms from my own experience, nevertheless, in the greater proportion of such children a certain neuropathic predisposition may be positively demonstrated. As the result of a long family practice I must emphasize the fact that some children who have suffered from eclamptic attacks and respiratory spasms during the first years of life have retained their tendency to convulsions until the age of puberty, although after the fourth year the respiratory spasms were replaced by convulsions arising in various disturbances of health, therefore by occasional spasms.

## V. TREATMENT

We have attempted to show that the convulsions of children are always the sequels of some other pathologic condition. Therefore, in many respects their treatment must coincide with the management of the underlying affection. In a *prophylactic respect* the prevention of infectious diseases, intoxications and injuries, on the one hand, and of rickets, on the other, is the best prophylaxis for the convulsive conditions of infancy.

First, a few general directions for the treatment of the spasm itself.

A child attacked by clonic convulsions, with loss of consciousness, must first be freed of its clothing and the respiration maintained by every means at our disposal.

If there is a suspicion of poisoning or evidence of an over-filling of the stomach, vomiting should be induced and eventually lavage of the stomach

should be practised. If the intestines are overloaded enemata should be given to empty the bowel.

*Hydrotherapy* is of special value in these attacks associated with loss of consciousness. During the convulsions, immaterial what their origin, the unconscious child should be placed in a lukewarm bath. While in the bath ablutions of cold water should be applied to the head and nape of the neck, or the head may be enveloped in cloths wrung from ice-water and changed at brief intervals. Small particles of ice may also be applied to the head and neck during the bath.

*Chloroform inhalations* are especially efficacious, and should be given in all prolonged convulsive states no matter what the age of the patient. The best method is to employ pure chloroform by the drop method.

Whether *compression of the carotids* is suitable in the treatment of convulsions of infancy I am not in a position to decide. At this time positive indications for this method of treatment are lacking, as the distribution of blood in the brain during the eclamptic attacks has not yet been determined. Accordingly there is no definite basis for judging the effect of this measure, which is of some value in the epileptic and uremic convulsions of later life. *Venesection* must be considered from a similar view-point. On the other hand, in long-continued attacks of spasm the application of *cutaneous irritants* is very serviceable in the form of mustard applied to the lower extremities, or of prolonged fresh mustard baths.

The remedial measures thus far mentioned are to be employed in persistent spasmodic attacks. The transitory eclamptic laryngospastic conditions which occur in rachitic children require energetic and rapid interference, for although the attack is of brief duration the child is in great danger in consequence of arrested respiration. Here the strongest cutaneous irritants must be applied at once. The child should be completely enveloped in cold cloths, or cold water should be applied with some force, by means of a syringe, to the sternum, the region of the heart, and along the vertebral column, to stimulate deep inspirations. The tongue must also be drawn forward and artificial respiration practised. If a battery is at hand faradization of the thorax should be employed.

During such attacks nothing is to be expected of drugs, not even of chloroform, which cannot be inhaled on account of the apnea.

In the consideration of *special therapy* for the convulsions of children the treatment of *occasional spasms* must be distinct from that of *hyperirritability convulsions*.

The treatment of occasional spasms must always be purely symptomatic. It is necessary to quiet the irritated nervous system by means of narcotics and to remove the underlying affection which gives rise to the convulsion. If the condition is the effect of foreign bodies, surgical measures are necessary; if due to a febrile disease the hyperthermia must be combated.

In regard to the *convulsions of fever* a cooling treatment by means of hydrotherapy prevents as well as aborts the attack in every febrile infectious dis-



case. For children who react readily with convulsions during febrile affections Raudnitz has proposed a prophylactic treatment for several months with small doses of the bromids, and states that he has seen good results from their use.

There are no specifics to abort the occasional spasms when once they have appeared. The treatment of spasms originating from the *intestine* coincides with the hygiene, the nutrition, and the therapy of infantile intestinal diseases. Rapid emptying of the bowel is necessary, and is best accomplished by suitable doses of *calomel*.

During the attacks of clonic spasm enemata of chloral ( $\frac{1}{4}$  gramme per dose), together with the internal administration of the bromids (1 gramme to 50 of water, a tablespoonful every hour), are particularly serviceable.

For prolonged convulsions tincture of musk may be employed (1 gramme to 50 of syrup, a teaspoonful every hour). The favorable effect of inhalations of chloroform in prolonged convulsions has already been remarked.

Regarding *myotonia* of the new-born and young nurslings a direct treatment is impossible. In the permanent spasms originating from the intestine a proper diet must be instituted; if the condition depends upon severe septic and cutaneous affections, these must be treated; if it results from syphilis, mercury must be administered. The form of myotonia dependent upon syphilis is particularly amenable to proper therapy.

The permanent spasms, dependent upon chronic autointoxication associated with atrophy, are most rapidly relieved by the infusion of normal salt solution, the simultaneous administration of small doses of calomel, and proper food. (See Figs. 11 and 12.)

In regard to the hyperirritability convulsions of children my remarks will be brief. Their treatment entirely coincides with the therapy of the underlying condition, *rickets*. *Treatment by phosphorus*, instituted by Kassowitz, is exceedingly effectual in such convulsions, as well as in the other nervous phenomena of early childhood dependent upon rickets, all of which yield readily to the influence of the drug. Most rapid to disappear is spasm of the glottis. Here phosphorus frequently has an actually miraculous effect; children who have suffered from spasm of the glottis several times daily for weeks are entirely relieved of this life-threatening laryngeal neurosis. The relief of the eclamptic attacks requires a longer time, and most protracted is the cure of tetany. It must, however, be emphasized distinctly that the disappearance of the spasmodic condition, which often occurs within a few weeks, must not interrupt the administration of phosphorus for the underlying affection, rickets, as the convulsions may very readily begin anew when this treatment is discontinued. Phosphorus should therefore be administered at least for several months. In severe eclamptic conditions there can be no objection to the administration of phosphorus at first in combination with the bromids, 5 or 6 teaspoonfuls of a 2 per cent. solution of sodium bromid being given daily during the first week in addition to a teaspoonful of a mixture of oil of phosphorus.

Phosphorus is most serviceable in the form of phosphorus cod-liver oil or phosphorus lipanin, or in an emulsion of oil as proposed by Kassowitz:

R.

Phosphori .....	0.01
Olei jecor. Aselli .....	100.0

M. et Sig. One teaspoonful daily.

R.

Phosphori .....	0.01
Lipaniini .....	100.0
Saccharin .....	0.1
Solve in pauxillo Alc. absol.,	
Ol. citri gutt. ....	III

Administered as above.

R.

Phosphori .....	0.01
Olei amygdalar. dulc. ....	30.0
Sacch. albi,	
Pulv. Gummi arabici, āā.....	15.0
Aq. dest. ....	40.0

Administered as above.

As all possible derangements of health, especially nutritive disturbances and diseases of the intestine, serve as exciting factors for attacks of spasm in hypersensitive children, a proper nutritive therapy is of the greatest importance, and as Finkelstein and Thiemich have demonstrated a direct dependence of electric contractility upon the manner of nourishment, much latitude is allowed this point in a therapeutic respect. Furthermore, as the etiology of hyperirritability convulsions is in intimate relation with rickets, although the latter affection is primarily dependent upon respiratory damage, fresh air, residence in airy rooms, ventilation of the nursery, and the avoidance of unpleasant odors are essential. Lastly, the prophylaxis of hyperirritability convulsions coincides with the general hygiene of infancy.

## VI. SUPPLEMENT: PATHOLOGIC ANATOMY

To refer to anatomical lesions in a functional neurosis is really a *contradictio in adjectis*. In construing the anatomical anomalies in the central nervous system which have been found in children who suffered from convulsions we must always remember that most of these children did not succumb to the convulsions but to some other simultaneous affection, so that in the individual case the first question to decide is whether the changes found in the nervous system can be brought into association with the simultaneous disease or the one

which has caused the spasm. This warning is especially necessary when an attempt is made to classify tetany as an organic disease of the central nervous system upon the basis of anatomical findings. Here a further complicating factor arises, the want of uniformity in the conception of tetany on the part of various authors who have reported anatomical findings in infantile tetany. Some anatomical lesions which have been found in children who succumbed to permanent spasms certainly have nothing in common with tetany; on the contrary, they belong to the realm of myotonia of nurslings, which I have separated from the category of tetany.

Again, where severe affections of a different nature (infectious diseases, chronic renal processes) were present, the findings do not permit of the conclusion that the changes found in the central nervous system bear any possible connection with the intravital spasmodic disease. In any event a definite anatomical basis can be construed neither for the occasional nor for the hyperirritability spasms of infants.

The lesions found in the central nervous system in children who suffered from spasm will be viewed differently if we take into consideration whether or not the attacks were of severe laryngospastic and apneic nature, and especially whether death occurred during an eclamptic-laryngospastic paroxysm. There can be no doubt that, as capillary hemorrhages occasionally occur in the cerebral cortex and in the meninges during attacks of pertussis, so also, in consequence of severe laryngospastic attacks, may hemorrhages appear in the central nervous system, which, however, are then not the cause but the consequence of the spasmodic condition.

The first anatomical findings in tetany were reported by Bonome and Cervesato.

In two children, aged one year and twenty-one months respectively, who had succumbed from febrile affections and were reported to have suffered from tetany, these authors found anomalies in the cervical enlargement and in the dorsal portion of the spinal cord. In the gray substance there was atrophy of the ganglion cells, increase of the neuroglia cells, here and there a diminution of the newly-formed neuroglia with the formation of irregular cavities. The lumbar cord and the peripheral nerves were exempt from change. These authors regarded the anatomical process as a poliomyelitis with predominant involvement of the ganglion cells of the anterior horns and a permeation of the process into the more deeply situated portions of the white substance.

Since we know that tetany, as such, does not give rise to a febrile disease, a severe infection in both cases is exceedingly likely, in the course of which a secondary poliomyelitis had arisen. It is also possible that these were in fact two rapidly fatal cases of acute poliomyelitis in children previously suffering from tetany. Both cases occurred in close succession in the Children's Clinic in Padua, and as the cumulative appearance of poliomyelitis in definite regions is a condition frequently observed, the lesions of the spinal cord demonstrated by Bonome and Cervesato may very properly be referred to epidemic poliomyelitis without any connection whatsoever with tetany.



The *black pigmentation* within the roots of the motor spinal and the cerebral nerves, stained by Marchi's method, which Zappert and, after him, Thiemich, E. Müller and Manicard have found in nurslings who had succumbed to a severe disease with spasms, has also been demonstrated in cases of tetany (Kirchgässer), but we now know that it is of no relevant importance in the solution of convulsions. In a case of infantile tetany R. Peters found a hemorrhagic pachymeningitis in the cervical and lumbar enlargements which was limited to the anterior and lateral portions of the periphery of the spinal cord, and continued to the coverings of the anterior roots as well as to the interstitial and connective tissues in the vertebral canal. In another instance he was able to demonstrate a neuritis of the motor roots, in a third case inflammatory changes within several spinal ganglia. All of these children, however, suffered from other severe disease (pneumonia) to which they succumbed.

Filia has examined the central nervous system of five children who suffered from hyperirritability convulsions, of whom one succumbed to pneumonia following scarlatina, two died of extensive atrophy, and two of laryngospasm. In one of the last two cases there were congenital changes in the spinal cord: a double central canal existed and the anterior of these canals was so distended as to suggest hydromyelia. In the cerebral cortex a deficiency in radial and tangential fibers was conspicuous. There were also microscopic findings in the cerebral cortex, in Roland's zone, especially thrombosis, foci of softening, and changes of the ganglion cells. The macroscopic investigation of the central nervous system revealed no pathologic change.

Carl Beck recently described two anatomical findings in children suffering from tetany. One, a child six years of age, who had suffered from visceral syphilis and contracted kidney in addition to typical tetany, showed, by means of Marchi's method, degenerative changes of the ganglion cells of the cerebrum, particularly of the motor cortical region, which could be recognized by an accumulation of fatty granular cells and granular globules, and further a fatty degeneration of the medullary fibers in the posterior roots of the spinal cord. The same changes were found in a boy aged one year, of whom it was only stated that he suffered from tetania gastrica. In both of these cases investigated by Beck the anterior roots were intact.

The lesions thus far reported in no wise justify the theory advocated by R. Peters that infantile tetany is an affection always dependent upon an organic disease of the spinal cord.

# DISEASES OF THE NOSE AND PHARYNX PECULIAR TO INFANCY (EXCLUDING TONSILLITIS)

BY J. ZAPPERT, VIENNA

## DISEASES OF THE NOSE

The cases of acute disease of the upper air passages which come under the observation of all practising physicians readily show that these affections occur in children much more commonly than in adults. Therefore, since coryza and pharyngitis are by no means rare in adults, the conclusion must be drawn that—aside from the great anxiety of parents for their children—these maladies are more severe in infancy than in later years and that in childhood certain affections are more frequent and often obtain a serious aspect. Chronic diseases of the nose and pharynx, which are more common in adults, are usually observed by the specialist rather than by the family physician, but in children's practice there are quite a number of cases which require a diagnosis by a general practitioner before they are recommended to the specialist.

The diseases which, either by their course or nature, are peculiar to infancy will be the subject of our present discussion, and so far as possible our attention will be devoted to them exclusively. We shall disregard, or merely mention in passing, those pathologic conditions of the nose and pharynx of which the symptomatology is alike in children and in adults and which are more peculiar to later youth. The *laryngological and rhinological methods of investigation* and their findings will not be comprehensively discussed, and are only mentioned in so far as they are indispensable for explanation. This omission is not intended as a disparagement of these noteworthy methods, but it is my experience that they are very difficult to employ in children's practice, and the younger the patient the more impracticable do they become, so that the physician is compelled to make a diagnosis without the aid of such measures.

Accordingly, the affections which will be considered in this article are limited to *acute rhinitis, involvement of the nose in infectious diseases, especially in syphilis, to epistaxis, foreign bodies in the nose, acute inflammations of the nasopharynx including Pfeiffer's glandular fever, to lymphadenitis retropharyngealis, retropharyngeal abscess, and adenoid vegetations in the nasopharynx*. Incidental remarks will also be made concerning other portions of infantile rhinology.

## ACUTE RHINITIS (CORYZA)

Children are no less exposed to *simple acute inflammation* of the nasal mucous membrane, *coryza*, than adults. The accompanying symptoms, however, vary according to the age of the child.

**Etiology.**—Among the causal factors *colds and contagion* are mentioned most frequently. We cannot discuss the question, in how far the influence of *temperature* is responsible for the development of a coryza, but we are not justified in ignoring this generally accepted view in our practical work. It is quite positive, however, that *infection* plays a much greater rôle in the development of coryza and is a particularly decisive factor in nurslings. In the case of children attacked by coryza who have not been out of doors for days or weeks previously, an inquiry concerning the health of the other inmates of the household, or an examination of them, will usually discover some member of the family or a servant who has been similarly attacked, and thus disclose the origin of the affection in the child. To know this and to express it forcibly is not superfluous from a practical standpoint. The anxious care of many mothers to prevent colds in their children will be effectually combated thereby, although not, perhaps, without the fatalistic assurance that protection of the child against catarrhal infection is almost impossible. Finally, the physician himself may be the occasional source of contagion in a child if he has carried on his children's practice during the period of his own rhinitis, often prolonged through several winter months.

Other causes which occasionally give rise to coryza in adults, such as the occupation inhalation of finely pulverized or toxic substances and drug intoxication, are of minor importance in children.

On the other hand, it is a well-known fact that *influenza* very often attacks quite young children, even nurslings, the initial symptom being a severe coryza. Although the rhinitis of influenza will be considered later somewhat in detail, it is necessary at this point to express a warning word in regard to attaching too much importance to influenza as a cause of coryza in children. Since the severe epidemic of influenza which passed over Europe in 1889-90 physicians are inclined to regard every acute febrile affection, associated with severe irritative phenomena and slight lesions of the respiratory apparatus, as influenza, and in the last few years it has been a quite regular occurrence, at the outbreak of a febrile coryza in children, to state that "influenza has again appeared." At variance with such a custom is the fact that any coryza may produce temperatures of 40° C. (104° F.) in children, to which Kohts called attention in 1878, therefore at a time when influenza as a pandemic was still unknown in Central Europe.

As yet we are unfamiliar with the *bacteriologic agent of coryza*. Chiari mentions as exciting factors the *asthmathos ciliaris*, *diplococcus coryzæ*, *pneumococcus*, *streptococcus*, *staphylococcus pyogenes albus*, *aureus*, *cereus*, *flavus*, etc. All of these microorganisms, however, have their habitat in the normal nose, therefore their pathogenic importance is limited.



Rhinitis is a disease of the *cold season*, occurring in young and old, and is never absent from December to April in families with many children. Also during the late spring and summer—especially after a drought—there is often a conspicuous accumulation of cases of rhinitis in children which is probably to be referred to a pathogenic agent which flourishes in heat and is readily transmitted by dust. A cause analogous to that of hay fever must also be taken into consideration.

**Symptoms.**—In the clinical picture of acute coryza, in young children particularly, the *hyperemic* may frequently be sharply differentiated from the *secretory stage*.

In such cases, in a previously healthy child, the disease begins with fever, rapidly rising to 40° C. (104° F.), associated with extreme lassitude. The patient is very restless or sleepless, and may even be somewhat somnolent. Older children complain of headache and thirst. This last symptom often occasions the remark from the mother that notwithstanding the fever the child is “quite hungry,” and forthwith overfeeds it with milk, as is shown by dyspeptic discharges. In older children it is easy to distinguish between a desire for much water and an aversion for all kinds of food. In uncomplicated coryza vomiting and convulsions are rare. In this stage of the disease objective examination reveals but little. The nasal mucous membrane is of a darker color and swollen, the posterior pharyngeal wall, exclusive of the tonsils and the palatine arches, is also hyperemic; otoscopic examination shows at most an injection of the tympanic membrane, without inflammatory phenomena.

These slight local symptoms may readily be the forerunners of all possible diseases of infancy, but the diagnosis becomes certain if, after a duration of one or several days of these initial symptoms, the second stage, that of *nasal secretion*, appears and the constitutional symptoms rapidly decline. There is frequent sneezing, at first with a serous, later a mucoid or mucopurulent secretion, sometimes noisy respiration, red, glistening skin of the nose especially in older children, and fissures and excoriations on the alæ of the nose, due to the flowing secretion which has not been carefully removed. In uncomplicated cases all of the disagreeable constitutional symptoms soon disappear; only the nasal secretion, which is at first profuse, remains and it also disappears gradually in the course of eight to fourteen days.

The catarrhal process is not always so simple, and in nurslings especially very unpleasant complications may arise.

In the child the *impermeability of the nose* is of great importance. Even in children beyond the age of nursing this symptom causes inconvenience, as they are often unable to blow the nose on account of the occlusion and cannot describe the seat and nature of the difficulty. From this inconvenience in the night an actual disturbance arises. Accustomed to nasal respiration, they do not readily learn to breathe through the mouth; therefore they sleep poorly and are awakened if the mouth should be closed for a time. The awakening often occurs with the symptoms of night terrors. The consequences of nasal occlusion in coryza may therefore resemble those of adenoid vegetations. Even more

annoying is the dryness and scratching in the throat, which, particularly after sleep, may interfere in swallowing and speech and cause tormenting cough. The dryness produced by mouth breathing, combined with the flow of mucus upon the posterior pharyngeal wall, produces a catarrhal irritation of the pharynx. Under some circumstances *in nurslings* this impermeability of the nose may become serious. Henoch describes, in a characteristic manner, the *dyspnea* due to nasal stenosis, which may lead to alarming attacks of asphyxia. For this sudden hindrance of respiration Bouchut offers the explanation that in consequence of enforced mouth breathing the tongue is drawn backward, the tip touching the hard palate, thus covering the entrance to the larynx.<sup>1</sup> In a desperate case the tip of the tongue must be held with a catgut suture and drawn forward. This acute asphyxia occurs more commonly during nursing—a time when an infant with nasal obstruction is forced to breathe strongly through the mouth. But even without such sudden attacks impermeability of the nose causes a decided *hindrance to the ingestion of food*. Upon closing the mouth the child becomes dyspneic, therefore the nipple of the breast or of the bottle is soon relinquished and often, after a brief combat between hunger and the fear of nursing, food is refused. It is clear that both of these factors, difficulty in respiration and in ingestion of food, may seriously affect the health, and that the prognosis of coryza is therefore more grave in infants than in older children. If fever and insomnia are also present, and if these symptoms last for a few days, the condition—aside from the possibility of sudden death due to nasal asphyxia—may actually become life-threatening. Death due to infantile rhinitis has been reported by West, Simon, v. Hüttenbrenner, and Baginsky. Kussmaul was able to save a child who was troubled by asphyxia during nursing by feeding for a week with the stomach tube.

*Other accompanying symptoms and sequels* of acute rhinitis are subordinate to those already mentioned. Thus the *impaired sense of smell* which is often so disturbing to adults is merely recognizable to older children, although the taste as well as the appetite of the child may also be unfavorably influenced.

*Excoriations* at the nasal orifices and upon the upper lip are much less common in infants than in children past the first year of life. Their absence in the former is probably due to the blowing of the nose, which prevents contact of the corroding secretion; and the spontaneous off-flow of the nasal mucus in infants, in consequence of their recumbent posture, occurs posteriorly rather than anteriorly. "Running nose" and excoriations are most frequently encountered in children who have learned to walk but who have not yet acquired the habit of cleansing the nose. There is a tendency of such catarrhal excoriations to become covered with yellow crust and thus assume the character of an *impetiginous eczema*.

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<sup>1</sup> The doubling up of the tongue, particularly when complicated by a long loose frenulum, may even lead to a "swallowing of the tongue," of which I have seen an instance. The tip of the tongue is drawn backwards and leads to instant suffocation. One of the famous German clinicians who died a few years ago told me twenty years ago of a baby of his own who died of the same accident.—EDITOR.

Among other sequels of infantile coryza its *transmission to the larynx and bronchi* is of serious import. In by far the majority of cases this propagation of the coryza is limited to the pharynx and larynx and is then for the most part characterized by a change in the timbre of the cough. The careful observer will sometimes note the transition from the toneless, scant cough with occluded nose to the tormenting, prolonged, staccato, pharyngeal cough which occurs principally in the morning, and to the rough barking tone of the laryngeal catarrh. The further extension of the catarrh into the bronchi and the lungs is by no means rare and depends often upon an influenzal character of the affection. Individual peculiarities undoubtedly play a prominent rôle. In some children coryza always remains localized in the nose; I know an anxious mother who proudly states that owing to her precautionary measures her child "never coughs when it has a cold." Much more common, however, are the cases of rhinitis which are almost regularly followed by implication of the larynx, sometimes associated with pseudo-croup. In such conditions we often learn that the father or the mother showed a similar tendency in their youth to a distribution of coryza, which would lead us to assume, as the cause for these sequels of the affection, hereditary anatomical anomalies of the nasal structure, and particularly adenoid vegetations, which recur in several generations.

The *distribution* of a coryza to the *accessory cavities of the nose*, on the one hand, and to the *ear*, on the other, must be variously considered. In a child, particularly an infant, the accessory nasal cavities are but slightly developed; the consequence of this anatomical peculiarity is an infrequency of disease of these regions in early infancy.

We know from the investigations of Kunkel (1873) that in the new-born the pharyngeal opening of the Eustachian tube is somewhat below the floor of the nasal cavity so that it is more readily surrounded by a descending secretion. On the other hand, it possesses but a slightly thickened margin, so that the opening has a more radiating form and is not funnel-shaped as in later life (Kohts). Therefore there is a plentiful opportunity for a transmission of the nasal catarrh to the ear, and as a matter of fact *catarrh of the tubes* and *otitis media* are among the most common accompanying conditions of infantile and especially of nursing coryza. It is the duty of every physician to bear this contingency in mind; he will then not be surprised, if there is a sudden rise of temperature in the course of coryza, and increasing restlessness and insomnia, that the case has already progressed beyond the first stage of coryza, and will think of a possible flow from the ear when epithelial masses in the auditory passage or a negative otoscopic investigation precludes a knowledge of the condition of the tympanic membrane. Even when there is no well-defined condition of the ear older children who are affected by coryza complain of "ear-ache," probably due to a slight catarrh of the Eustachian tube. Not rarely the primary symptoms of coryza are overlooked by the parents or neglected. The restlessness and symptoms of pain are referred to colic and the physician is not called until discharge from the ear appears. There are, however, epidem-



ics of nasal catarrh, with a special tendency to ear affection, in which the initial nasal symptoms are so slightly developed as to give the impression of a primary otitis. Rey describes epidemics of this apparently primary middle ear catarrh, and I also have observed that at certain periods nearly all the children in my practice who were affected by coryza showed aural affection, while at other times very much severer forms of rhinitis ran their course without implication of the ear.

In a review of the factors which characterize coryza of children, and especially of nurslings, in contrast with that of adults, we note the following: *The abrupt onset with marked febrile phenomena, the slower transition into the stage of secretion, the important consequences of nasal occlusion as regards sleep and the ingestion of food, the possibility of asphyxia, the rare but dangerous conditions of exhaustion, the frequency of pseudocroup and the great tendency to affection of the ear.*

It scarcely requires mention that, in addition to those children who present a great proportion of these conditions when attacked by coryza, there is a predominant majority of little patients to whom all of the symptoms are absent, the coryza running just as harmless a course as in adults.

The transition of simple coryza into the *chronic catarrh* is not common in children in the absence of other predisposing factors. Such factors are furnished especially by adenoid vegetations, scrofula and syphilis. We are then not dealing with simple rhinitis but with the sequel of other diseases which will be considered under their proper headings. It is only necessary to mention that coryza which frequently relapses should always awaken a suspicion of adenoid vegetations, and in this sense often requires treatment.

Thus far we have considered simple acute coryza—rhinitis *par excellence*—which, in most cases at least, must be regarded as a substantive infectious disease whose pathogenic agent is still unknown and the particular localization of which is in the nose. The nasal mucous membrane, however, is also involved in the majority of *acute infectious diseases*, either as a first point of attack or secondarily.

I. It has been indicated in this article that even in infancy *influenza* may primarily attack the nose and produce severe symptoms. The diagnosis between simple coryza and that due to influenza is not always easy and is often dependent solely upon secondary symptoms and sequels. If there is a case of influenza in the family a febrile coryza in a child at once arouses suspicion, particularly as infants—inversely to other infectious diseases—are exceedingly susceptible to influenza. As a rule the fever and constitutional symptoms are severer in influenza than in ordinary coryza, and convulsions at the onset are not rare. The nasal secretion, especially in older children, soon becomes purulent and is often very profuse. The demonstration of Pfeiffer's influenza bacillus in the nasal mucus is not very difficult and naturally is the only positive proof of a correct diagnosis. In favor of influenza is the rapid propagation of the disease to the deeper respiratory passages and the development of severe otitis with a hemorrhagico-purulent exudate, as well as the occasional forma-

tion of vesicles upon the discolored, dark red, tympanic membrane. An uncommon but very serious complication of influenza is disease of the meninges, which occurs as a secondary infection. Disease of the auxiliary cavities in children in general is rare, as has already been mentioned, but its occurrence in later childhood must be considered. Neuralgia, muscular pains, and general lassitude cannot be demonstrated in nurslings but these conditions occur in older children just as in adults. From these facts, therefore, it is evident that the coryza of influenza is a common and by no means insignificant affection in children and also in infants.

II. It has been remarked that the cumulative attacks of coryza in older children in the early, hot summer days, present a condition analogous to *hay fever*. Contrary to this hypothetical conception, however, is the fact that in unquestionable epidemics of hay fever children are just as frequently attacked as adults and develop the same symptoms of the affection (Phöbus-Blackley).

III. In *measles* the nasal mucous membrane is always affected early, and after a maculate redness, soon shows diffuse hyperemia and swelling. The nasal symptoms are not very severe; sneezing and secretion are usually only present in the stage of onset. Sometimes a swelling of the mucosa and a tendency to chronic coryza remain. That the pathogenic agent, so long searched for without result, is present in the nasal secretion of measles is made exceedingly likely by Mayr's old inoculation experiments. The frequency with which disease of the ear and of the deeper respiratory passages occurs in measles is well known.

IV. *Rötheln* (rubella) usually runs its course with slight involvement of the mucous membrane. The implication of the nasal mucosa is correspondingly slight, although according to Pospischill it is never entirely absent.

V. In *erythema infectiosum*, which has lately been much discussed, the absence of initial rhinitis has frequently been emphasized. Judging from my experience this view, as well as that of an exemption of the pharyngeal mucous membrane, which is often maintained, must in time be corrected, the latter condition not being characteristic of the affection.

VI. Coryza does not belong to the initial symptoms of *scarlatina*; in contrast to the foregoing affections disease of the nose is secondary. On the other hand, purulent coryza appears as an accompaniment of necrotic processes in the throat. This was regarded by Rilliet and Barthez as a serious sign and it deserves this appellation in full measure, although not so much on account of the local affection as because of the severe pharyngeal disease of which it is usually an associate. The condition runs its course with decided swelling of the nasal mucosa and considerable muco-purulent secretion, causing an extension of the inflammation of the mucous membrane to the nasopharyngeal space and pharynx and a decided hindrance of nasal respiration. In debilitated, feverish, septic children the disturbance of sleep and the deficient inhalation of oxygen resulting from this condition are by no means of slight importance. The dyspneic attacks of "pharyngeal stenosis," due to occlusion of the nose and pharynx, which resemble laryngeal croup, are well known, but a differentiation is possi-

ble through the absence of laryngeal cough and the predominant involvement of the nasopharynx. In such cases cleansing of the nose by means of pledgets of cotton, and—as affection of the ear is already present in this stage of scarlatina, or is very likely—irrigation of the nose with antiseptic fluids, are of vital importance and often bring about decided amelioration. For this purpose LeLorier introduces a soft urethral catheter with lateral openings into the nose, leaving the free end outside the mouth. To avoid corrosion by the secretion around the nasal orifices the frequent application of lanolin, borated vaselin, zinc ointment, etc., is advisable.

VII. In *diphtheria* disease of the nose plays an important rôle. In infants especially that organ may be the primary and perhaps the only seat of the diphtheritic infection, or is secondarily involved in a distributed pharyngeal process. *Primary nasal diphtheria* in children, even in those but a few days old, is markedly infectious and is justly feared in foundling asylums. Usually the onset of the affection is insidious, beginning with the flow of fluid mucus; often in this stage only the unilateral appearance, in contrast to ordinary coryza, is suspicious (Hüttenbrenner). The secretion gradually assumes a hemorrhagico-purulent character, the nasal openings become excoriated and not infrequently are covered with diphtheritic ulcers; the deep cervical glands enlarge, the temperature shows a decided elevation, the child appears to be severely ill, becomes somnolent, and occasionally, but not invariably, particles of membrane are found adherent to the walls of the nose or to the cotton pledgets, which clears the diagnosis. Sometimes there is a propagation to the pharynx and larynx. As a rule death soon releases the sufferer. Nasal diphtheria in nurslings therefore represents a conspicuous septic form of the malady.

Less dangerous, but nevertheless serious, is nasal diphtheria as a secondary phenomenon of disease of the larynx. This form also is at first characterized by a mucoid secretion which soon changes to a purulent or hemorrhagic composition. Excoriations, ulcerations, edematous swellings form at the nasal orifices just as in the variety last described. As a rule, enlargement of the lymph glands is associated with this transmission of the diphtheritic process to the nose, but this is not alone due to an inflammation of the nasal mucous membrane but is an expression of the *septic character of the diphtheria, and diphtheria of the nose may be regarded as its signum*.<sup>1</sup> Although these are the

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<sup>1</sup>The observation that “glandular swelling is not alone due to the nasal mucous membrane, but may be the expression of a septic character of diphtheria” may permit of some modification. The worst cases of septic diphtheria are those of mixed origin. The enormous glandular swellings are less due to the diphtheric bacillus or toxin, than to copious infection. Indeed, uncomplicated diphtheria exhibits no big glandular swellings, and then only when it is nasal. The worst cases of septic diphtheria, when not of a mixed character, are frequently not attended with much swelling. They result from a direct absorption from the (eroded and bleeding) bacillus and toxin into the blood circulation, circumventing the lymph apparatus altogether. In those terrible cases death can be averted only by very frequent mildly antiseptic irrigations of the nares and by large doses of diluted alcohol and other stimulants and antiseptics internally.—EDITOR.



signs of a severe course of the disease the prognosis is not so unfavorable as in the foregoing variety.

A form of nasal disease designated RHINITIS FIBRINOSA or RHINITIS PSEUDOMEMBRANACEA has been much discussed among pediatricists. This affection is characterized by a pathologic picture developing with quite harmless symptoms of occlusion of the nose and dryness of the pharynx, which may continue for weeks or even months, when suddenly—often to the great surprise of the physician—a membranous coating appears in the nose. A case reported by Henoch, occurring in a daughter of Traube's, is celebrated and characteristic. This girl, aged eight years, was attacked with coryza, moderate fever, decided snoring while asleep, and complained of a hindrance in respiration near the root of the nose. The pharynx and epiglottis revealed catarrhal redness. After a duration of several days the child expelled a tough white membrane of about the length of a finger from the nose, followed later by a smaller one of similar composition. A few days afterward the child was well. A longer duration, with a more gradual discharge of particles of croupous membrane is the rule. There is always absence of membrane in the pharynx as well as of severe general symptoms. The disease is scarcely infectious, although it occasionally appears in several children of the same family. Is this rhinitis fibrinosa, or pseudomembranacea as it has been called, true diphtheria? The condition was for a long time obscure. At first only cocci were found, principally pneumococci (Abel); the bacillus of diphtheria was not demonstrated (v. Stark). In later investigations, however, the diphtheritic organism was found with such regularity (Baginsky, Concetti, Stamm, Abbot, Luigi, Scheinmann, Treitel and Koppel, Buys, Morf, Neumann, and others) as to leave no doubt of the true nature of the affection. Post-diphtheritic paralyses also have been reported after rhinitis fibrinosa (Luigi). From these reports it may be concluded with certainty that this form of inflammation of the nasal mucosa is simply an attenuation of a diphtheritic disease and that Spirig is right in rejecting the name rhinitis fibrinosa and substituting for it the term *rhinitis diphtheritica*. Naturally, in all cases of this kind the employment of serum is necessary in the treatment.

VIII. In *varicella* eruptions appear upon the nasal mucous membrane, especially where it is in contact with the external skin, as well as on the pharynx and conjunctivæ.

IX. *Gonorrheal rhinitis*, although rare, must be regarded as an assured condition. The original opinion that the gonococcus cannot thrive upon the nasal mucous membrane has been proved fallacious by the investigations of Finger, Ghon, and Schlagenhauser. Primary gonorrheal disease of the nose in the new-born is to be placed in the same category as gonorrheal conjunctivitis and, like that affection, is due to contamination by the infectious vaginal secretion of the mother (Weber, Fraenkel, Stoerk, Ziem, Bresgen, etc.). According to Stoerk it has been maintained by Leopold Müller that a secondary infection of the nose from the diseased conjunctivæ is possible and is even frequent; he has invariably found gonococci in the nasal mucus of infants affected

with *blennorrhoea neonatorum*. Although this finding is not synonymous with nasal gonorrhea, nevertheless, in children with such affections of the eye, the nose must be carefully watched and upon the appearance of a secretion of pus antiseptic or astringent solutions must be employed.

**Treatment.**—From the discussion of the symptomatology of infantile coryza it is evident that the treatment covers all of the gradations from purely expectant to urgent, life-saving measures.<sup>1</sup>

In older children in whom the coryza runs its course without special disturbance treatment is not necessary, but we must bear in mind the possibility of complications. If the initial phenomena are severe an attempt may be made to abort the attack, i. e., to bring about secretion in the first stage, that of fever and hyperemia. The child is put to bed, and a diaphoretic tea is given (lime blossoms, elder flowers, hot lemonade), perhaps with the addition of a few decigrams of *sodium salicylate*. Filatow, who does not regard sodium salicylate very highly, advises in “endemic grippe,” which he differentiates from “epidemic grippe” or influenza, *muriate* or *sulphate of quinin*, either in the form of powder, 0.06 for each year of life, or in solution. Instead of quinin analogous preparations, such as aristochin, euchinin, or, when fever and headache are prominent, antipyrin or salipyrin. An old method of treatment of coryza consists of *hot foot baths*; of less use in children’s practice are sinapisms to the back, employed by the older physicians. As a rule, in small children and especially in nurslings, all of these measures should be dispensed with in the febrile stage of onset, the treatment consisting merely of *cold compresses* to the head, “bathing with vinegar,” and *cold packs*.

Whether older children with well-developed coryza are to be treated is a matter of preference of the physician or of the parents. The number of remedies is much too large and great reliance should not be placed upon any one of them. The local measures consist of inhalants, the insufflation of powders, applications, spraying or mechanical cleansing of the nose.

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<sup>1</sup> The large number of recommendations proves the difficulty of treatment. The nares and post nasal space must be left clean. The mildest application is salt water 7:1000. Cotton pledgets soaked in it should be used frequently. Warm irrigations should be made frequently, hourly or in longer intervals, from a small Whitall Tatum, Birmingham, Dessar or other cup, care being taken that the current reaches the throat. Adrenalin (1:1000) could be applied by means of the pledget. Cocain is not so good because the secondary hyperemia is great. Astringent solutions (zinc. sulph., zinc. sulpho-carbol., alum) should be quite mild. In occasional cases the difficulties are great. I have been compelled to use the actual cautery on the luxuriant mucous membrane for days in succession, and to keep the mouth open constantly to keep the baby from suffocating. We had no adrenalin then which might have rendered the electro-cautery unnecessary.

The difficulties of ozena are by no means overdrawn. Irrigations should be made at least twice a day; a few days with “saline solutions” to accustom the patients to the procedure; then with permanganate of potassium 1:3000-4000. Once every few weeks I use a saturated solution of silver nitrate. Before applying it the lips and orifice of the nose must be moistened with a fairly strong salt solution. The application should be made with cotton wrapped round a straight whalebone or silver probe long enough to reach from the nares to the pharynx. A spoonful of salt solution should be swallowed immediately for obvious reasons.—EDITOR.

Preparations of *menthol* are preferable for *inhalation*, and lately have been combined with formaldehyd in the form of *forman cotton*. Also serviceable are camphor, oil of turpentine, ammonia (Hager-Brand remedy: acid. carbol., liq. ammon. caust., āā 5.0, spirit. 15.0, aq. dest. 10.0; Sig. Inhalations every two or three hours from the bottle or a few drops poured on thick blotting paper and held to the nose [Kiesselbach] ). In children's practice, however, these and many other inhalants are unsuitable, for unless they are breathed very carefully with closed mouth they readily produce cough and burning sensations in the eyes and frighten the child by their pungent odor. Under such circumstances a repetition of the inhalation often meets with great resistance. Furthermore, the success of these inhalants is problematic, and if their result is limited to sneezing it may be attained in some other manner.

*Insufflation of pulverized substances* by means of an insufflator may be practised with children provided they remain quiet. Here, in addition to the astringent-antiseptic effect, sneezing and expulsion of the stagnant nasal secretion are of no trifling importance. Among the remedies employed for this purpose are sodium sozoiodol, boracic acid, tannic acid, and alum. The various powders which contain morphia or opium should not be employed in children.

In infants our purpose will best be attained by *applications* to the mucous membrane of the nose, which requires but slight skill with a small brush or with a wooden applicator covered with cotton. Such a cleansing of the nose is necessary in nurslings because of the difficulty in the ingestion of food. For this purpose watery or glycerized solutions of boracic acid (2 per cent.), silver nitrate ( $1\frac{1}{2}$  per cent.), protargol (3—5 per cent.), sulphate of zinc (0.5 per cent.), alum (10 per cent.), or cocaine (2 per cent.) will prove of service, or if crusts have formed, salves are preferable, either yellow precipitate, boracic acid or zinc ointment, vaselin with menthol, olive oil, etc. Even when the intranasal application of salves is not employed, moistening of the nasal orifices with these ointments, glycerin, or cold cream, is desirable.

In all of these local measures more importance is to be attached to a thorough cleansing of the nose than to an antiseptic action of the remedies. Kiesselbach's process—the introduction of cotton pledgets with a bent forceps or with a spiral tampon carrier, which is turned further by the hand and left in place for fifteen minutes—should be considered in the coryza of nurslings.

Finally, *washing of the nose*, for which a number of implements are in use. The employment of all instruments presupposes a willingness on the part of the patient, with which we cannot reckon in infant practice, as crying and irregular respiration not only make the value of these nasal washings very illusory but may also cause injury to the ear. This form of treatment for coryza has therefore been somewhat neglected in children.

In the local treatment of the nose we must not overlook the sequels of coryza, which are often more important than the primary disease. Particular attention must be given to the difficulty in sucking. The nose should be cleansed several times daily, perhaps before each feeding, with a plug of cotton. If the breast or the bottle is not well taken the food must be given with a spoon. In cases



of this kind breast-fed children must be nourished with milk pumped from the breast. If sufficient food is not taken at one time it must be administered more frequently. Stubborn cases of rejection of food render necessary feeding by means of the stomach-tube. Some authors advise cocainization of the nasal mucous membrane, even in nurslings, when food is rejected in consequence of coryza; for example, Nägeli-Ackerblom introduce a drop of a solution containing cocain 2.0, aq. dest. and glycerin āā 50.0 into each nasal opening three or four times a day by means of a dropper.

An affection which differs from ordinary rhinitis and requires a separate description is

### SCROFULA OF THE NOSE<sup>1</sup>

Scrofula is the collective term for all chronic processes which appear in small children upon the mucous membrane of the upper respiratory passages, in the glands, and in the skin. The nosologic conception of this term is now more uncertain than ever and the identification of scrofula and tuberculosis is still general among pediatricists (Henoch, Biedert, Monti, Soltmann). This condition of affairs would account for Spitzmüller's recent definition of scrofula: By scrofula we understand that change in metabolism, and particularly in the circulation of the lymph, whereby a pathologic predisposition arises in the organism to infect itself with pyogenic bacteria and the tubercle bacillus. With this indefinite classification of scrofula it is clear that the affection of the nose also can only be defined with difficulty, and whether he shall diagnosticate a simple chronic inflammation or scrofula in the individual case very often devolves upon the personal view of the physician.

In recent text-books upon rhinology (Chiari, Bresgen, and others) scrofula of the nose is not considered as a substantive affection but is included with the various forms of chronic rhinitis.

If we intend clinically to adhere to scrofula of the nose, the condition may be regarded as such when there are no recognizable external causes for the existing pathologic picture (adenoid vegetations, foreign bodies), and it must therefore be assumed that the changes present are the expression of a constitutional disease.

**Pathology.**—Nasal scrofula is a part phenomenon of the "torpid" form of the affection, to which it gives the characteristic impress. The condition is one of hypertrophic rhinitis in which the nasal mucous membrane is swollen and reddened, the lower turbinated bone usually enlarged; not rarely there is loss of epithelium, and ulceration of the nasal mucous membrane. A constant and disagreeable symptom is the accumulation of a profuse mucopurulent secretion within the nose, leading to occlusion, as well as to eczema, fissures, excoriations and ulcers at the outlet of the nasal passages.

These lesions upon the external skin are very tenacious. Exceedingly often they are the cause of inflammation and stasis of lymph in the skin of the

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<sup>1</sup> See article on "Scrofula," this volume.

nasal alæ and of the upper lip, which are then characteristically swollen and reddened. After this chronic-purulent stage of rhinitis has lasted some time there appears almost always a swelling of the lymph glands under the sternocleidomastoid and along the deep jugular vein, which become very large and even suppurate. Occlusion of the nose leads to alteration in speech, which assumes a characteristic "nasal twang" (Baginsky), and to attacks of sneezing and coughing. Chronic catarrh of the pharynx and larynx is a frequent result of rhinitis. Purulent affections of the middle ear as well as lachrymation and catarrh of the conjunctiva, due to occlusion of the tear ducts, are not infrequent.

Quite a different picture is presented by atrophic rhinitis, *ozena*. Here the mucous membrane is in a stage of involution, the turbinated bones are small and atrophic, and in consequence the nose is wide, the secretions scant and formed into crusts, and, above all, the odor from the nose is unbearable. A scientific controversy exists as to whether *ozena* belongs to hereditary syphilis and scrofula or only to the first-named affection. Without attempting to prejudice a decision we shall refer *ozena* to syphilitic disease of the nose.

**Prognosis.**—Naturally the prognosis of scrofulous rhinitis cannot be separated from that of the underlying affection. It is not particularly unfavorable, although we must not forget that in a moderate number of cases swelling of the bronchial glands and other signs of tuberculosis may appear in addition to scrofula. The nasal symptoms, however, are prone to improve, and when we note the vast number of children between three and fourteen years of age with scrofulous rhinitis, and the small number of adults with this nasal affection, we must admit that the trouble very often disappears. *Ozena* is of unfavorable import and almost incurable.

**Treatment.**—The treatment consists primarily in combating the scrofulous constitution, and the internal administration of iron and cod-liver oil. Iodin and sea baths, mountain climate and favorable nutritive conditions are of particular value (see Soltmann's article on Scrofula and Tuberculosis, this volume). Locally, medicated bougies, insufflations of powder, applications and sprays are to be employed.

Following Catti, Chiari advises nasal bougies consisting of small gelatin cones of tannin (0.1), copper sulphate (0.02), silver nitrate (0.1), potassium iodid (0.1), etc. Solutions of silver nitrate (2-10 per cent.), tannin, glycerin and iodine, creolin vasogen (2 per cent.) (Strübing), eucalyptol (ol. eucal. fol. 1.5, spir. vini 20.0 and aqua 200.0, Baginsky), etc., are serviceable for application.

For a spray or for nasal washings normal salt solution, potassium permanganate, boric acid, etc., may be employed. Particular attention should be devoted to careful cleansing of the external nose, removal of crusts, application of ointments, and eventually the cauterization of ulcers or of the moist eczema. A process that is worthy of mention is that of Witthauer, who after loosening the crusts in the nose, cauterizes with a 2 per cent. silver nitrate solution or with a solution of iron chloride, and every evening introduces cotton tampons

dipped in glycerin and water and thickly covered with powdered alum (Baginsky). In severe cases we must resort to surgical treatment of the hypertrophied turbinated bones.

## HEREDITARY SYPHILIS OF THE NOSE

The nose is a point of predilection for hereditary lues; in fact no other part of the organism shows such early and permanent signs of the malady. For clinical purposes it is best to divide the affection into an *early* and a *late form*, but that these are not so sharply separated as might appear will become evident later.

**Symptoms.**—The characteristic *early form* of lues is *coryza syphilitica*. This prevails in an overwhelming majority of infants who are hereditarily syphilitic and in those cases in which symptoms appear in the first months of life it is probably never absent. It is frequently a forerunner of the luetic exanthem and is sometimes the only sign of infantile syphilis. *Coryza syphilitica* is often congenital (among 63 cases investigated for such evidence by Hochsinger it was found 38 times), or becomes manifest in from four to six weeks. The syphilitic coryza of nurslings in its characteristic form is a *rhinitis hyperplastica*. The mucous membrane is greatly swollen and injected, and the secretion, at least at the onset, is quite scant. As the swelling principally involves the lower turbinated bones the interior of the nose is greatly narrowed. In general in recent cases the diffuse inflammatory process is limited to the anterior portions of the nose.

The narrowness of the internal nares is the primary cause of the clinical symptoms.

A very characteristic factor of syphilis of the nose is the *snuffling* during breathing and is usually the first symptom which brings the child to the physician.

Even in this stage, the nasal opening is often found excoriated with fissures; fever is never present in uncomplicated syphilitic coryza.

In cases that are rapidly and energetically treated syphilitic coryza may be arrested in this stage and, for a time at least, give rise to no further trouble.

More commonly the condition occurs in connection with the secretory stage. The secretion is never very profuse but causes corrosion of the skin and a purulent or even hemorrhagico-purulent discharge. The respiration now assumes a more sluggish character, and sometimes, although not commonly, sneezing occurs.

From limitation of the nasal respiration dyspnea arises as well as difficulty in the ingestion of food. The first-named symptom is rarely so marked as in acute rhinitis. The latter, however, may lead to decided disturbance, analogous to that described under acute coryza.

The excoriations upon the outer surface of the nose are quite conspicuous; there may be crustaceous ulcers at the nasal orifices and upon the upper lip



which, by occluding the nares, naturally increases the difficulty of nasal respiration.

In severe untreated cases, in addition to syphilitic coryza an ulcerative necrotic stage may occur which may lead to deformity of the nose. We shall recur to this condition later.

Hereditary nasal syphilis may also be the cause of disturbances in the immediate vicinity of the nose, but it never gives rise to bronchitis and pneumonia, and in this respect it differs from coryza simplex of the new-born.

The **prognosis** of the early form of nasal syphilis is accordingly not unimportant. Aside from the immediate disturbance in consequence of hindered nourishment, the condition may result in further changes in the nose which will be discussed as late symptoms of hereditary lues.

Even with the early institution of antisyphilitic treatment the course is chronic and insidious. Failure to recognize the condition and negligence in treatment may readily lead to far-reaching local disturbances.

The **treatment** coincides with that of general syphilis and needs no special explanation. Local treatment of the nose is usually unnecessary. If it appears desirable inunction or painting with a  $\frac{1}{2}$ -1 per cent. solution of corrosive sublimate is of service.<sup>1</sup>

Syphilis does not confine itself to this early stage; there may be changes in the nose which do not appear until the *close of infancy or even later* and which must be regarded as an expression of *syphilis hereditaria tarda*. We owe a minute knowledge of these late forms of nasal syphilis to Fournier, and Gerber has contributed an interesting report of this condition. In adhering to Fournier's classification and terminology we shall not enter into a discussion of his conception of *syphilis hereditaria tarda* as a late manifestation of a previously unrecognized or recognized hereditary syphilis, which may perhaps require complement. It is a fact that as a rule the late forms of hereditary nasal syphilis appear unexpectedly toward the end of infancy and even later, without obvious precursors of such a condition.

**Ozena.**—A form of nasal affection which is sometimes encountered in the subjects of hereditary syphilis is *ozena* (*rhinitis chronica atrophicans or atrophica*, Chiari). Among rhinologists this affection has been much studied and greatly discussed. The etiology especially has been the subject of comprehensive researches, and in addition to a number of bacterial findings in *ozena* the relation of the disease to syphilis and scrofula has frequently been investigated. Thus Stoerk, an authority of unquestioned importance, is of the opinion that all cases of *ozena* are the late results of syphilis.

To decide or investigate this point is not the function of the pediatricist. So far as we are concerned it is certain that *ozena often develops in individuals hereditarily syphilitic*, whether or not this constitutional disease is the only basis of the affection. Their connection has been proven, or at least considered very likely by a great number of investigators (Stoerk, Gerber, Hochsinger,

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<sup>1</sup> The application of corrosive sublimate  $\frac{1}{2}$  to 200 seems too strong.—EDITOR.

Fournier, etc.). Syphilitic ozena is predominantly an affection of late childhood and puberty or, more correctly, it usually becomes manifest at this stage of life. Unfortunately, the onset is very insidious, characterized neither by pain nor other obvious symptoms, and the affection usually presents itself to the physician in full development.

The anatomical lesion of ozena consists in an atrophy of the mucous membrane, of the cartilages, and of the turbinated bones, so that the interior nares appear dilated. The secretion is not profuse, and is comprised less of mucus than of pus and serous fluid; it is very tenacious, adhering firmly to the walls of the nose, where it forms crusts and decomposes. From this decomposition, perhaps under the influence of specific microorganisms, a dreadful odor emanates which, although less noticeable to the patient himself than to those about him, makes the affection exceedingly mortifying. In children this affliction may result in an actual inhibition of normal development, as it renders contact with other persons impossible and prevents attendance at school or the learning of an occupation. Such persons are greatly restricted in their opportunities for maintenance and are much to be pitied. Ozena is perhaps the most cruel legacy which a syphilitic father can leave to his child. In some cases it is possible, by rinsing the nose, to uncover decomposed ulcers or even portions of sequestered bone; sometimes the patients are able to remove small particles of bones from the nose by blowing. This, according to Chiari, is a noteworthy differentio-diagnostic factor of syphilis in contrast to ordinary ozena, in which bone necrosis does not occur.

Sometimes, after the removal of these particles of bone and antisypilitic and antiseptic treatment, it is possible to cure ozena. In the majority of cases, however, the prognosis is very unfavorable and Stoerk's pessimistic view of the affection is only too correct.

Convincing as is the theory of this author of a relationship between hereditary syphilis and ozena, the therapeutic test of this view is of little consequence: the antiluetic treatment is absolutely ineffectual—a result, however, which is also frequently noted in the late forms of syphilis. The local treatment of syphilitic ozena likewise offers but slight hope of success. The internal administration of iodine, antiseptic washings, insufflation of powder, inunctions, vibration massage, curettement, electrolytic cauterization, as well as injections of paraffin, lately practised, for the formation of a lower turbinated bone and extension of the surface of the mucous membrane (Fliess), have been attempted with varying results.

In addition to ozena, *marked changes in the shape of the nose* occur as the consequence of hereditary syphilis. The fact that these deformities, which are without question associated with syphilis, are often combined with ozena—in Gerber's experience, in 43.3 per cent. of his cases of ozena—should also serve to substantiate the syphilitic nature of the latter.

The nasal deformities which are recognized by Fournier and Hochsinger are as follows:

A common change of the nose results in simple *pug-nose*; the dorsum of

the nose is somewhat depressed and the root broadened, so that the nostrils are forced forward and upward.

A second important deformity results from complete destruction of the external structure of the nose, whereby "the tip of the nose, after loss of the cutaneous covering, adheres to the upper lip" (Hochsinger's *Bocknase*).

A very characteristic and well known form of hereditary syphilis is the *saddle-nose*: Here the depression of the dorsum is so marked that in profile it sinks deeply under the frontal bone, the tip of the nose being drawn upward as though torsion had been made upon the bridge. This nasal deformity is quite common; to the expert it is the unmistakable sign of syphilitic taint in the youthful bearer.

Less common are two other deformities of the nose, the knowledge of which we owe to Fournier.

One is a lateral depression designated the *spectacle nose* (*Pincenez* or *Kneifernase*), in which the forepart of the nose is somewhat broad and on both sides of the dorsum it appears depressed, corresponding to a nose upon which eye-glasses have long been worn.

The second deformity is the so-called *lorgnette nose*, which is probably better designated the *telescope nose*. The anterior soft part of the nose is forced into the posterior bony portion, like the parts of an open telescope. This produces a fold of skin over the forepart of the nose at the point where it sinks into the posterior portion. If the tip of the nose is pulled forward and downward the deformity disappears and the organ assumes its normal contour.

How do these manifold deformities of the nose in hereditarily syphilitic individuals originate? For some of the cases which are found in lues hereditaria tarda there is no satisfactory history. As Fournier has remarked, they have no prior pathologic history but have developed gradually or have existed so long that the patient has forgotten their onset. In other cases necrotic processes are present in the bone, mostly affecting the vomer, the lower turbinated bone and the ethmoid bone (Fournier). According to Gerber such a necrosis may occur in the hyperplastic stage of nasal syphilis in small children and early produce the nasal changes which are regarded as a late form of syphilis. But destruction of the internal nares may appear even in nurslings, although, as emphasized by Hochsinger, they then involve only the cartilaginous and cutaneous portions of the nasal structure.

Hochsinger furnishes some very important material for the study of this condition. Among 256 infants with hereditary syphilis he found changes in the nasal structure in 62, nearly all of which conformed to the deformities mentioned above; also in a few cases there was perforation of the nasal septum, and a child aged seven months showed perforation of the hard palate. This destruction was always associated with a purulent or hemorrhagico-purulent discharge from the nose; therefore Hochsinger designates this form of nasal syphilis as a stage of exulceration, that is, a deformity associated with the simple hyperplastic or suppurative stage of coryza syphilitica.

If all of these findings are borne in mind those cases of nasal deformity



which are met with as the expression of lues hereditaria tarda, without a plausible prior history, lose to some extent their strangeness. We can probably assume that these are the permanent results of an early syphilitic nasal affection which was treated insufficiently or perhaps not at all. A sharp distinction between early and late symptoms of nasal affection in hereditary syphilis, as has been frequently made, then disappears and it need not be surprising that a constitutional disease which shows a predilection for the nose in intrauterine life as well as in early infancy eventually leaves in this structure unmistakable and permanent consequences.

### FOREIGN BODIES IN THE NOSE

The presence of foreign bodies in the nose occurs in children much more commonly than in adults. This is explained by the tendency of children to introduce small, smooth objects into the nose in play, which remain there readily on account of the narrowness of the orifices and the insufficient power of expulsion. It is also rare for the child to designate the cause of his nasal trouble, so that the physician must of himself determine the presence of a foreign body. Reasons for such a suspicion are offered by *unilateral* occlusion of the nose, sneezing, altered voice, headache, painfulness and swelling of the nose. If the foreign body has been present in the nose for some days there is usually a purulent, sometimes *bilateral*, nasal catarrh which gradually assumes a fetid character, leading to corrosion of the skin at the entrance of the nose; in this stage confusion with nasal diphtheria is possible. The catarrhal inflammation of the nasal mucous membrane may be propagated posteriorly, affecting the Eustachian tube and with this the ear. Under some circumstances foreign bodies may remain in the nose for months or even years without being recognized.

The foreign substance is sometimes discovered immediately upon inspection of the nose or after it has been cleansed. In other cases this is more difficult, especially where there has been time for reactive phenomena. Touching of the nasal mucosa with a solution of cocain and adrenalin is a valuable aid as it causes constriction and anemia of the membrane.

The *foreign bodies that are encountered in children* are various; beads, stones, buttons, beans, peas, cherry-seeds, and pellets of cotton are the most common. The most unpleasant substances are those which swell in the nose, such as beans and peas; Boyer observed a case in which a pea introduced into a nostril had begun to sprout. In small children, particularly in infants, vomited masses are sometimes discharged through the nose; such an occurrence may also happen in diphtheritic paralysis during deglutition. As a rule such masses pass quickly through the nose, but a retention of firm particles is possible. They may then decompose and lead to irritation of the nasal mucous membrane. A special form of foreign substance in the nose is represented by *animal parasites*. Aside from the cases in which caterpillars, flies, earwigs, ascarides,

oxyuris, etc., gain entrance to the nose it is necessary to consider the severe inflammatory phenomena which arise in that structure and in the auxiliary cavities from the larvæ of flies. In temperate climates these occurrences are very rare, and then are often only secondary infections in somnolent children with decided nasal secretion; in tropical regions, however, there is a disease due to the larvæ of flies (*myiasis*, *Peiper*<sup>1</sup>), associated with severe symptoms, which leads to destruction of the internal nose and, by giving rise to meningitic phenomena, may even terminate fatally.

At this point the relation of the *boring of the nose to intestinal parasites* must be briefly considered. The laity see a reliable symptom therein and bring their children to the physician for this phenomenon alone for medicine to expel the worms. Naturally, we must reject such an idea and negative a connection of worms with nasal boring. Perhaps this as well as so many other popular views and superstitions may have arisen from a misunderstanding of some former medical experience, as that the transmission of the ova of the oxyuris by means of the finger into the nasal passage causes itching, which is plausible.

**Treatment.**—The treatment of foreign bodies in the nose consists in their removal. In mild cases strong blowing of the nose during occlusion of the free side will prove effectual; in other cases nasal washing, which, however, must be avoided if objects which are liable to swell are suspected. If these measures are insufficient to expel the substance an instrument must be used—a Daviel spoon introduced behind the obstacle to force it forward. More hazardous is the manipulation with a forceps, by which means however round flat substances are readily forced backward. This should be avoided, because the foreign body may become attached in the nasopharyngeal space or find its way into the larynx and trachea. In older children painting with a cocain-adrenalin solution prior to the operative interference is advisable. Ulcers due to pressure require antiseptic treatment, which is best accomplished by the insufflation of powder (sodium sozoiodol, dermatol, boric acid, etc.). If the foreign body is so embedded that it cannot be removed *per vias naturales*, operative interference by division of the nose must be considered.

Injections of chloroform-water (Chiari), alum, carbolic acid, alcohol, and turpentine, insufflations of tobacco smoke and fumes of ether for the removal of animal parasites have been practised with success.

## BLEEDING FROM THE NOSE (EPISTAXIS)

*Epistaxis* in infants is rare. This may be due to absence of causative constitutional conditions, to the fact that the mucous membrane of the nose is less succulent and rich in blood, or to the lesser liability to excoriations from pick-

<sup>1</sup> See volume on "Diseases of Metabolism of the Blood and Animal Parasites and Toxicology," p. 573, *et seq.*

ing and scratching of the nose. It must not be overlooked that in the nursing in the recumbent posture bleeding from the back of the nose, as well as from the nasopharynx, is more apt to occur posteriorly than anteriorly, so that even if epistaxis is present it need not be apparent. These considerations are of practical importance, for Swoboda has found that in children with *melenae neonatorum* the passage of blood through the intestine is due to hemorrhage from the nose. In his cases there was also epistaxis, but Hochsinger reports a case in which bleeding of the nose *ante partum* led to the discharge of hemorrhagic masses from the intestine, and not until an inspection of the pharynx was suggested by Swoboda's publication was epistaxis discovered; after tamponage of the nose the hemorrhage was arrested. These cases of nose-bleeding in the new-born, however, are rare. More common is a bloody admixture of the nasal secretion in chronic diseases of the nose, as in coryza syphilitica.

Epistaxis in older children, as in adults, is due to *trauma, constitutional conditions, or local changes in the nose* ("spontaneous" epistaxis).

Strictly speaking, most forms of epistaxis must be considered as *traumatic*, since it is usually some external cause which leads to injury of a nasal blood-vessel and to hemorrhage. Here, however, we will consider as traumatic epistaxis only those forms which are due exclusively to external agency. This usually consists of a blow or fall upon the nose. In severe accidents it is also necessary to consider fracture at the base of the skull. Aside from possible deeper injuries of the nasal structure these traumatic hemorrhages as a rule neither diagnostically nor therapeutically give rise to great difficulty; more disagreeable are the external injuries to the nose.

Few of the *constitutional conditions*, which in adults often lead to nasal hemorrhage, are common to childhood. Even in valvular disease of the heart, nephritis, and pulmonary affections, in which stasis occurs, epistaxis is rare in infancy. The condition is most frequently seen in acute endocarditis, of which diagnostically it is often a valuable premonitory symptom. Hemophilia may produce severe nose-bleeding in children and is often recognized by this symptom. Epistaxis also occurs in purpura, scurvy, etc., and less frequently in Barlow's disease. As an accompanying symptom of general infectious diseases of childhood (measles, scarlatina) it is not so rare and in *whooping-cough* it is often important and serious. In the last affection, however, the blood which flows from the nose as well as from the mouth does not always originate from the nasal cavity, but from vessels located elsewhere which are ruptured in the severe paroxysms of cough associated with great cyanosis. In pertussis this hemorrhage is usually not prolonged, but when once it appears it may be repeated frequently during the acme of the disease.

Epistaxis of *older school-children* requires special consideration. Baginsky quite correctly asserts that this may be regarded as a school disease, due to confinement in the close air of the schoolroom together with the simultaneous mental exertion, and quotes Kotelmann, who found epistaxis in the higher grades of school much more frequently than in the lower (28.33 per cent.



against 12.16 per cent.). A likewise unquestioned, although insufficiently explained, fact is the epistaxis of boys and girls at *the time of puberty*. In girls this bleeding sometimes appears with the menstrual period and is sometimes said to compensate for it. Stoerk believes that young men with nasal hemorrhage have not practised coitus but suffer from sexual irritation. That there is a connection between the nose and the genital organs is unquestioned. How far epistaxis can be regarded as a sign of this relationship is a subject worthy of discussion but no longer belongs within the scope of infantile nasal diseases.

The so-called *spontaneous epistaxis*, as has recently been proven (Michel, Kiesselbach, Chiari, and others) usually originates in an area of the cartilaginous septum which is so far forward that "the patient may reach it with his finger." Dust and other external agents cause itching, and excoriations are produced by the finger which lead to the rupture of a superficial vessel. The loosely formed thrombosis is readily dislodged by slight causes, but even without an obvious cause a further hemorrhage may occur. If the nose is examined with a speculum the suspected area is readily recognized by dilated vessels in the vestibulum nasi at the anterior end of the quadrangular cartilage (Mihalkowicz, Chiari); rubbing with a pellet of cotton is often sufficient to start the hemorrhage anew. Under some circumstances deep ulceration may occur at this point and even perforation of the septum.

**Symptoms.**—The symptoms of epistaxis require no special description. The blood usually exudes by drops, rarely in a stream. As a rule the quantity is exaggerated by the laity. Nevertheless, pallor, vertigo, and syncope are by no means rare, although the peculiar "fear of blood" plays a rôle in many children and such symptoms should not immediately be attributed to anemia of the brain from blood loss. The blood which flows along the posterior pharyngeal wall is often swallowed in considerable amounts and may afterward be vomited. The possibility of vomiting or the evacuation of masses tinged with blood after severe epistaxis should be mentioned to the parents. On the other hand, it should not be forgotten that in apparently spontaneous hemoptysis and hematemesis—also in nurslings with hemorrhagic discharges (see above)—the possibility of epistaxis is to be considered.

**Prognosis.**—The prognosis of epistaxis in childhood is usually good. As a rule the loss in strength from profuse flow of blood is soon regained.

**Treatment.**—The arrest of the hemorrhage and the prevention of its recurrence constitute the treatment of epistaxis.

The first duty of the physician, when called to a case of nose-bleeding, is to quiet the frightened child and to convince the family, who perhaps are running about with sponges, vinegar, cologne water, etc., of the harmlessness of the condition. At the same time a brief examination of the pulse and the appearance of the patient assures him of the patient's general condition. With the child comfortably seated, or in a recumbent posture if syncope is threatened, the constricting collar and clothes are removed, the child is induced to breathe deeply and to restrain from blowing of the nose and coughing, and Bresgen ad-

vises also the avoidance of expectoration on account of the transitory stasis. To the child's care-takers is allotted the task of placing cold compresses upon the nose, the forehead, and the nape of the neck. In the meantime the physician has determined from which side of the nose the hemorrhage originates, for which a careful cleansing of the accessible parts is necessary. A thick tampon of cotton is then placed in the nostril and pressed against the septum. This alone is often sufficient to control the hemorrhage. If, following this provisional tamponage, blood appears from the other nasal opening or around or through the cotton plug, a piece of ice is placed into the bleeding cavity and enclosed with cotton. Hot water is equally of service. If this treatment is ineffectual, styptics may be employed, of which, however, only the Penghawar Djambi still possesses advocates. Sesqui-chlorid of iron which was at one time much employed has lost somewhat in favor on account of the possibility of corrosion, but might be tried, for a brief time only, in the commercial form of 30 per cent. iron chlorid cotton. Recently ferropyrin cotton has been strongly recommended (Lublinski, Breshen). More valuable than these styptics is the suprarenal substance in the form of adrenalin (adrenalin hydrochlorid, 0.1 to 100 of water) and other combinations. Of this solution one part to nine of water is dropped upon cotton and pressed against the bleeding surface. Instead of water 2-5 per cent. of cocain or eucain may be employed, which not only increases the hemostatic effect of the adrenalin but also produces an agreeable sensation of relief.

Less successful than the application of styptics with simultaneous compression by means of cotton is cleansing of the nose with hemostatic solutions. This procedure may easily defeat the contraction of the vessels as the fluid removes the thrombus which may have formed.

As a rule these measures will prove successful, but we must not omit an examination of the posterior pharyngeal wall to determine that the blood which has been stopped anteriorly by a tampon is not flowing off behind. If such should be the case, or blood soon reappears anteriorly, complete tamponage of the bleeding side of the nose must be performed. For this purpose iodoform gauze, or, better, iodoform-tannin or iodoform-collodion gauze, should be employed, as the continuance of such tampons *in situ* for several days will prevent the decomposition of blood and mucus. Airol gauze is also recommended (Hanszel). The introduction is accomplished by a nose or ear forceps, a dressing forceps or grooved director. An attempt must be made to reach the choana so as to completely fill the nasal cavity. Chiari advises (for adults) three or four pieces of gauze 20 cm. in length and of the breadth of two fingers, folded in the middle, one end of which is carefully introduced into the nostril until it is firmly lodged in the bony structure of the nose. The remaining portion of the gauze is then folded in until the nostril is completely occluded. This tampon is kept in place one day and the coating of nasal mucus as a rule permits of its easy removal.

As a last resort, in severe epistaxis tamponage by means of Belloq's tube, or a catheter or drainage tube may be employed. The tube is introduced through

the nostrils and pharyngeal space into the mouth, where a tampon, corresponding in thickness to the patient's thumb, is fastened to the end and anchored in the choana. It is necessary to adjust it in position by the finger. This tampon is fastened anteriorly by means of the thread with which it was attached to the sliding instrument; some rhinologists fasten it in addition by a second thread which passes through the mouth. After clogging of the choana, tamponage of the anterior nasal cavity must be performed. This method is disagreeable from the time it is instituted until removal of the tampon and should if possible be avoided in children.

In hemorrhages which are not so severe that a minute inspection of the bleeding area is impossible, rhinologists now cauterize at once, thus combining arrest of the bleeding with a *permanent cure* of the epistaxis. This method is necessary in those relapsing cases of spontaneous epistaxis which originate from the area previously mentioned upon the anterior surface of the nasal septum. Cauterization is best practised with solid chromic acid or the galvano-cautery after anesthetization by cocain or cocain-adrenalin. Substances with a strong odor, such as trichloroacetic acid or fuming nitric acid, had better be avoided in the child as they cause restlessness even before their introduction into the nose. The nose must not be blown after the cauterization and the cotton tampon must remain for one day. One or two applications are often sufficient for a permanent cure of the trouble.

In the *general treatment* of children with a tendency to epistaxis a possible underlying affection, prevention of a secondary anemia, the avoidance of excessive bodily and mental exertion, are of importance. School-children who suffer from frequent nose-bleeding should be kept from school for some time and perhaps altogether. Nose-bleeders should be cautioned against picking and scratching of the nose and the disagreeable habit should be overcome before unpleasant consequences arise.

## DISEASES OF THE NASOPHARYNX

In accordance with the plan formulated at the beginning of this article this closes the discussion of the nasal diseases of infancy. There are other affections of the nose, as neoplasms, injuries, tuberculosis, cutaneous diseases, but none of these conditions is peculiar to infancy and their course is not different from that in adults. Also deformities and anomalies of the nose, especially of the septum, although congenital, are usually matters for later consideration and treatment, requiring the attention of a specialist, and thus are remote from the province of the general practitioner.

We shall therefore pass to a discussion of diseases of the *nasopharynx*, which will be considered from the same standpoint as the pathologic conditions of the nose.



## ACUTE INFLAMMATION OF THE NASOPHARYNX; PFEIFFER'S GLANDULAR FEVER

The maladies of the nasopharynx have a greater importance in infancy than at any other period. It is probable that the richness in lymphoid tissue of this region, that is, the ease with which swelling arises, which is peculiar to infancy, is the principal reason why these affections are mostly observed by the pediatricist.

Nevertheless the acute affections of the nasopharynx have not as yet been distinctly defined and occasionally a symptom-complex is presented which is not easy to diagnosticate.

The difficulty of a proper recognition of such inflammatory affections in childhood, particularly in infancy, is enhanced by the awkwardness of local examination of the posterior nares; therefore it is obvious that together with the common and manifold inflammations of the *pharyngeal structure*, the frequency of *acute diseases of the nasopharynx* removes a large number of infantile affections from direct inspection, so that often merely a tentative diagnosis can be made.

In view of these difficulties it seems proper to call attention to a diagnostic aid which is of value not only in the recognition but also in the localization of nasopharyngeal affections: *acute swelling of the lymphatic glands of the neck*.<sup>1</sup>

The pharyngo-oral and pharyngo-nasal cavities are not only permeated by a profuse lymphoid tissue, known as Waldeyer's "tonsillar ring," but also by an extensive plexus of lymph-vessels. The lymphoid or adenoid tissue is found in great aggregations, forming the "pharyngeal tonsil," yet to be described, in the nasopharynx as well as in the tonsils, and is also present in the mucous membrane, for example, at the opening of the Eustachian tube. On the one hand, these adenoid elements form a suitable receptacle for infection and a common seat of inflammatory processes; on the other hand, the numerous lymphatic vessels cause a rapid absorption of irritative products and their conduction into the adjacent lymphatic glands, which react with painful swelling. This rapid inflammatory involvement of the lymphatic glands at their root areas is a phenomenon peculiar to infancy, the cause of which has not been positively determined. But the child does not react alike to all inflammatory affections of the pharyngeal structure; the rapidity, intensity, and firmness of the lymphatic gland enlargement depends greatly upon the degree as well as the nature of the primary infection (for example, the characteristic hard lymph nodules in diphtheria). *In many cases intumescence of the lymph glands does not occur, but when it does arise we may expect a certain orderly arrangement whereby definite glands corresponding to every diseased portion of the nasopharynx are always the first to enlarge.* The knowledge of the relation of the individual regions of the nose and pharynx to the regional lymph nodules is

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<sup>1</sup> Trautmann (*Jahrbuch f. Kinderhk.*, 60, Bd. III) gives a comprehensive description of these conditions.

therefore not to be under-estimated, and offers valuable diagnostic points of support.

The important results obtained by anatomical investigation we owe—aside from the older reports of Mascagni, Sappey, and others—principally to Henle, Stahr, and Most.

We learn therefrom that the region of the pharyngeal tonsil, the tonsils, and the base of the tongue possess a particularly profuse plexus of lymphatic vessels. In the nose the lower turbinated bones, the floor of the nasal cavity, and the choanæ possess numerous lymph-vessels which become more scant in the septum, and are markedly decreased toward the entrance of the nose as well as in the upper turbinated bones and in the roof of the nose. The *glandulae cervicales profundae superiores* (Henle) are to be regarded as the essential outflow station of the lymphatic vessels from the pharynx and nasopharynx. These are situated at the point of bifurcation of the external and internal carotid arteries and follow the jugular vein to the base of the skull; a *well-defined lymph nodule* is situated in the triangle formed by the jugular vein, the facial vein, and the lateral belly of the biventer muscle. Another group of deep cervical glands is situated laterally to the jugular vein upon the posterior scaleni muscle and the cervical plexus, and partially upon the posterior portion of the sternocleidomastoid. This important portion of the deep cervical gland group is designated by Most the *glandulae cervicales profundae laterales*; for the posterior portion the name *gland of the nape of the neck* is employed. In addition, at about the height of the soft palate, on the boundary between the posterior and lateral walls of the pharynx and in front of the lateral portion of the rectus capitis there is one lymph gland, rarely more, which probably is in relation with the deep cervical glands just mentioned, but which deserves especial consideration on account of its constant situation. Most, who has called particular attention to it, has named it the *pharyngeal gland*. An inconstant accumulation of small but never numerous nodules is met with upon the posterior pharyngeal wall; these are designated *glandulae retropharyngeales*. Most regards these merely as a passage to the pharyngeal and cervical lymph nodules. The *submaxillary glands* (eight to ten in number, Henle) are situated upon the inner surface of the lower jaw. The nodules situated between the anterior bellies of the biventer muscle upon the chin are designated the *submental glands*.<sup>1</sup>

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<sup>1</sup> The rôle of the tonsil in causing adenitis and ready absorption is overestimated. These effects are mainly due to the function of "Waldeyer's lymphatic ring," and the accompanying pharyngitis. The lymph communication of the tonsil with the neighborhood is limited; affection of the tonsil when localized has very little influence in causing neighboring adenitis. When inflammation, or diphtheria of the tonsil extends beyond it, to the neighborhood, secondary adenitis will result to a higher degree. The finding of tubercle bacilli, diphtheria bacilli and cocci in the lacunae of the tonsil is no proof of their being absorbed and dangerous, just as little as the presence of bacilli and cocci in the nose means tuberculosis, or diphtheria, or toxemia. Microbes found in the tonsil are very apt not to be absorbed, for good anatomical reasons, but to be expelled again on the road they came on. [Arch. of Pediatrics, June, 1907.—EDITOR.]

*In what relation are these glands to the region of the lymphatic vessels of the different portions of the pharynx and of the posterior nasal cavity?*

The *post-pharyngeal* lymph-vessels as well as those of the *lateral and upper portions of the pharynx* permeate the pharyngeal wall, first touching the retro-pharyngeal glands, mentioned above, and then the *glandulae pharyngeales* (Most). Thence they pass to the *deep cervical glands* (*glandulae cervicales profundus superiores*), to which also some vessels pass directly. A portion of these lymph-vessels, and particularly those which originate from the *upper portion of the nasopharynx* and vault of the pharynx, pass directly to the *lateral cervical glands* (*glandulae cervicales profundus laterales*), avoiding the pharyngeal gland. The lymphatic vessels of the *tonsils* and *palatine arches* pass immediately to the *deep cervical glands* and especially to *that nodule*, mentioned above, which is situated in the *triangle* between the jugular and lateral facial veins and the biventer muscle. The lymph-vessels of the lower portion of the *pharynx* and the *region of the larynx* (Most) as well as of the upper portion of the *thyroid gland* (Bartels) have their stations in the *glandulae cervicales profundae superiores*.

Supplementarily a few other root regions of lymph glands upon the neck will be mentioned.

The lymphatic vessels from the *anterior portion of the nose* and the *septum* empty in part into the facial gland situated upon the parotid, partly into the submaxillary gland. The lymph from the *interior nose* makes its way posteriorly through the choanæ and thence in part directly to the deep cervical gland, in part—conjointly with the lymph-vessels of the posterior pharyngeal wall—first to the *glandulae pharyngeales*. The lymph-vessels of the *buccal mucous membrane* (Polya and Navratil), as well as those of the mucous membrane of the *upper jaw*, empty largely into the submaxillary gland; only a small portion pass directly to the deep cervical gland. Upon the *lower jaw* one portion passes to the submaxillary gland, another to the submental gland. The *skin of the cheeks* has its lymph station in the parotid, submental, and partly also in the submaxillary glands. The *lips* (Dorendorf) empty their submucous lymph-vessels into the submaxillary gland, their subcutaneous lymph-vessels partly (upper lip) into the submaxillary gland and partly (lower lip) into the submental lymph gland. From the *tongue* the lymph tracts pass into the submaxillary gland, the *glandulae cervicales profundae*, and the *glandulae mentales* (Küttner, Jordan, Porier).

The following are the results of this anatomical presentation, which are of practical importance as regards the pathologic enlargement of the glands:

Swelling of the *submaxillary gland*, which is not found at the angle of the jaw but upon the inner side of the lower jaw, arises from disease of the buccal mucous membrane, the gum of the lower jaw, the upper lip and the tongue, or the skin of the cheek, but not from disease of the pharynx. In enlargement of the *submental gland* an affection of the mucous membrane of the lower jaw, as well as of the lower lip, is to be considered. If the *pharyngeal glands* are enlarged, disease of the posterior pharyngeal wall, principally of the



middle area, as well as of a portion of the internal nose, may be assumed. On the other hand, in affections of the *upper portion of the pharynx*, therefore also of the *nasopharynx*, the *lateral deep cervical glands* (including the glands of the nape of the neck) will be found enlarged, and may be felt below and behind the sternocleidomastoid muscle. In inflammation of the *tonsils* and of the *palatine arch* an enlarged nodule will be discovered at the bifurcation of the facial and jugular veins, in front of the sternocleidomastoid muscle, at about the height of the hyoid bone. The deep cervical glands will also be enlarged and may readily be felt in the furrow in front of the sternocleidomastoid and below it. The last-named glands will be found intumescent in severe inflammatory processes in the *deeper regions of the pharynx*, in the *laryngeal area* as well as in the upper portions of the thyroid gland. *In addition they are the terminal stations of a number of other lymph-gland regions of the neck and will be found enlarged in all inflammatory processes of the nasopharynx and the pharynx.*

*A knowledge of these facts permits us to form an opinion from the acutely enlarged lymph-glands as to the seat of the primary affection and therefore contributes valuable auxiliary aid in the recognition of acute inflammations of the upper air passages.*

After this excursion into the anatomical precincts, which I do not believe to be superfluous, the clinical aspect must be considered, and first in order are the *acute inflammations of the nasopharynx*.

A differentiation may be made between *artificial and idiopathic inflammation*:

*Artificial infections of the nasopharynx*, although rare, are the consequence of operative removal of adenoid vegetations. As a rule this operation is not followed by secondary phenomena, that is the process of healing remains strictly local, as is usually seen also after tonsillotomy. In rare cases, however, there are apparently deep-seated inflammatory processes which, in their final result, are mostly harmless, but which present a quite characteristic picture. The most prominent symptom of this secondary infection is painfulness of the cervical region, which enforces a rigid fixation of the head (Thost). In passive movements of the head there is a lively counteraction similar to the condition in meningitic children with rigidity of the neck. This painful tension of the neck musculature, however, more closely resembles muscular rheumatism, especially when it is unaccompanied by fever. The early objective examinations of such children show but little. Inspection of the pharynx merely reveals a downward flow of mucus along the posterior wall of the pharynx, as is common after adenoid operations, and in one of my cases even posterior rhinoscopy and digital examination of the nasopharynx by an expert gave a negative result. Careful palpation of the nape of the neck is liable to reveal the condition earliest, as we can thus determine whether the bellies of the muscles are less painful than the furrows between them. This palpation is also of decided value if we are able to demonstrate an enlargement of small pearl-like glands of the neck behind the sternocleidomastoid.

It has been indicated that the enlargement of these glands in particular signifies an affection of the nasopharynx, and this enlargement, therefore, in conjunction with the history, may be regarded as positive evidence of an inflammatory infection following operation. In the case quoted above there was a sudden discharge of pus from the nose from a region which could not be detected by rhinoscopy, from which the child soon recovered. Moderate fever was present during the prevalence of the lymph-gland enlargement.

Notwithstanding its rarity, this form of post-operative inflammation of the nasopharynx has been considered in detail because it aids our understanding of the *idiopathic inflammation* of this region.

This disease asserts itself as follows: A child formerly healthy, or who has previously shown a similar condition, is suddenly attacked by high fever ( $40^{\circ}$  C.- $104^{\circ}$  F.), pain in the limbs, vomiting and headache, as in follicular tonsillitis. The tongue is coated and there is complete loss of appetite. The early examination reveals but little and inspection of the pharynx especially is unsatisfactory.

It is true, the posterior pharyngeal wall, if it can be examined at all, appears inflamed, but the tonsils and palatine arches are normal or are so slightly injected that they cannot be the source of the pyrexia. Speech is sometimes conspicuously nasal, as in occluded nose, but there is neither impermeability nor coryza, nor does the otoscopic examination reveal anything of importance. The tympanic membrane is either normal or moderately injected.

This obscure and disturbing condition may continue for some days without change; often, however, a new symptom is added, which is not calculated to reassure the parents. The child complains of pain in the throat, localized, if it is sufficiently intelligent, to the nape of the neck, not as the pain of swallowing. As a matter of fact the neck is sensitive to pressure and its movability is hindered. Palpation of the neck, especially of the nape, now often shows positive findings: Along the posterior border of the sternocleidomastoid muscle, on one or both sides, are found a number of small, painful glands, corresponding to the glandulae cervicales profundae laterales and the glands of the nape of the neck. Simultaneously, or soon afterward, enlargement of the deep cervical glands below the belly of the sternocleidomastoid is noted. With enlargement of these glands in many cases the acme of the disease is reached and the fever declines rapidly. Sometimes, however, there are relapses, the temperature remaining high. The glands increase in extent and size, but final recovery may be confidently expected. Suppuration of the glands does not occur.

To the expert it is clear that this is the clinical picture of a disease known as **Pfeiffer's glandular fever**, which has been much discussed in pediatric literature. Pfeiffer, in his description, has especially mentioned the enlargement of the glands at the nape of the neck *behind the sternocleidomastoid, resembling a rosary*, as a characteristic feature of the affection, and says literally: "Also the glands situated anteriorly to the sternocleidomastoid are often involved in the process, but their enlargement and painfulness is not character-

istic, as they are enlarged and painful in many other affections, for example, in tonsillitis, stomatitis, etc."

Heubner, however, in a supplement which he contributed to Pfeiffer's original communication, has extended this view by including cases in which "the lymph glands situated below the sternocleidomastoid" greatly enlarge, with fever and a rigidity of the head, without any demonstrable affection in the mouth, pharynx, or elsewhere. In subsequent reports regarding this affection a sharp distinction is not made between the acutely swelling glands, and Hochsinger, whose views coincide in the main with those given here, in a reference to Pfeiffer's publication, speaks particularly of an enlargement of the "cervical or submaxillary" lymph-nodules.

From the foregoing presentation of the relations of the lymphatic glands of the neck it seems absolutely desirable to limit the pathologic picture, somewhat more sharply than was previously done, to *enlargement of the lateral cervical glands*, and to regard this as a *sign of disease of the upper part of the pharynx*. In the further course of the affection the deep cervical glands also enlarge, but never the submaxillary lymph-nodules.

The above clinical picture, which corresponds with the original description of Pfeiffer's glandular fever, may therefore be referred directly to an *acute inflammation of the nasopharynx*, and coincides also with that form of disease which proceeds from a post-operative inflammation in this region.

We have not considered the rôle played by *adenoid pharyngeal vegetations*—the so-called *Luschka's tonsil*—in the occurrence of these acute inflammations of the nasopharynx, as it is desirable to adhere chiefly to the localization of diseases in the pharyngonasal cavity. There can be no doubt, however, that such an inflammatory process without involvement of the pharyngeal tonsil is improbable and that acute inflammation of the retronasal space principally represents an inflammation of adenoid vegetations.

This is favored by the anatomical relations, by the tendency of the lymphatic tissue to inflammation and to retention of mucus and secretion, and, finally, by clinical experience. The last teaches that children with an enlarged pharyngeal tonsil not only show a particular tendency to inflammation, in the form of Pfeiffer's glandular fever, but that in such children relapses of this acute disease of the retronasal mucous membrane form the rule, and that removal of the adenoid growths often brings about permanent recovery.

These *relapsing inflammations of the nasopharynx* are often worse than relapsing tonsillitis and are an actual plague to patient and physician, as it is impossible for the latter to demonstrate the true seat of the disease to the parents of the child, and his diagnosis is therefore readily mistrusted. These children may have four, five, and even more, attacks of puzzling fever in a winter and may also markedly emaciate from the accompanying loss of appetite. In relapsing retronasal inflammation the glands of the nape of the neck as well as the cervical glands are enlarged. All of these considerations justify the view, expressed above, that *the clinical picture, under the name of Pfeiffer's glandular fever, represents an inflammation of the pharyngeal tonsil*. On this



point we agree implicitly with Hochsinger, who, some years since, promulgated the same opinion in a lecture.

*What are the causes of this acute inflammation of the nasopharynx?* As we are here dealing principally with a disease characterized by its localization, it is likely that different causes may bring about the same pathologic result. One of these—post-operative infection—has already been mentioned. Scarlatina might also produce a like pathologic picture, which may give rise to diagnostic perplexity if it appears during the time of defervescence. It is exceedingly likely that influenza is also an etiologic factor for this form of inflammation. Finally—and on this great stress must be laid—retronsal acute inflammation, or Pfeiffer's glandular fever, so often appears cumulatively that it immediately gives the impression of a substantive infectious disease. Pfeiffer emphasizes this cumulative appearance of the affection—even in house epidemics—and for this reason considers it a nosologic entity, distinct from other diseases of the glands of the neck. So far as is known there are no reports of bacteriologic investigation but it is not unlikely that the bacillus of influenza is responsible for the entire condition.<sup>1</sup>

Finally, a few words regarding the *varieties* of acute retronsal inflammation.

Pfeiffer and other more recent authors have demonstrated that the transitory fevers of children, the "ephemera" or "febricula," to which, as "Fièvre éphémère," a special chapter in the *Traité des Maladies* is devoted, are often abrupt forms of these retronsal inflammations, which seems quite plausible.

In contrast thereto are the curious general infections following glandular fever, which run their course with enlargement of the spleen, liver, and retroperitoneal lymph glands, and intestinal disturbance. Heubner has also mentioned nephritis as a severe complication of the disease. We will not enter upon a detailed description of these perplexing clinical cases. Several conditions which appear to me to be unrelated to the affection have been included, and, as has already been indicated, the clinical picture of retronsal inflammation can be produced by various underlying affections. Perhaps the relation between the lymphatic pharyngeal ring and the lymphoid tissue of the vermiform appendix, which has lately been disclosed, will render more intelligible some further enigmatic complications of "Pfeiffer's glandular fever."

From the foregoing, however, we recognize in the acute inflammations of the nasopharynx, particularly when occasioned by adenoid vegetations, an important affection of infancy, a knowledge of which is of great value to the physician, who otherwise is liable to commit many diagnostic errors. Professional skill is usually of little avail, particularly as local treatment of the child is almost impossible; in retronsal inflammations of this kind we are limited to general remedies for control of the fever, compresses to the neck, frequent cleansing of the nose, applications of astringent antiseptic solutions to the posterior pharyngeal wall by way of the nostrils. The treatment of this affection

<sup>1</sup> Trautmann (*Jahrbuch f. Kinderhk.*, 60, Bd. III) in one case demonstrated streptococci.

coincides in the main with that of adenoid vegetations, and, in the relapsing forms particularly, there should be no delay in the removal of such proliferations.

Having considered acute inflammations of the nasopharynx it would now be proper to discuss the *chronic inflammatory processes* of this region.

Clinical considerations, however, seem to make it more proper to include the chronic inflammations of the pharyngonasal cavity in the description of adenoid pharyngeal vegetations. The latter not only constitute the most common and positive cause of chronic nasopharyngeal disease but one is an anatomical transition of the other, so that they can scarcely be separated clinically; it is usually difficult to determine where the signs of simple "adenoids" terminate and those of the associated catarrh begin. Chronic inflammation of the nasopharynx without hypertrophy of the adenoid tissue is such an accidental occurrence in practice that it may be left out of consideration altogether. We shall therefore pass to a discussion of

### ADENOID VEGETATIONS IN THE NASOPHARYNX

If a historian were to chronicle the events of medicine from the standpoint of the momentous changes which a single personage has often brought about in the various realms of science he would scarcely find a more characteristic example than the development of our knowledge of adenoid vegetations of the nasopharynx.

Existing for centuries, distributed over all parts of the world, described, as regards their consequences, by shrewd investigators, but not themselves recognized, this form of disease was completely revealed and almost exhaustively described through the faculty of observation of a single man, and soon became an exceedingly fertile field of labor for the medical practitioner, forming today a valuable permanent asset of professional knowledge and activity.

Dupuytren had previously described a pathologic picture consisting of occlusion of the pharynx and evinced surprise that tonsillotomy did not bring about a cure. Czernak, the inventor of the laryngoscope, gave a rhinoscopic description of the proliferations without entering into the subject further. Voltolini even performed an operation for adenoids which was successful.

Other authors also had occasionally referred to anomalies in this region, but to a Danish otologist, Dr. William Meyer of Copenhagen, belongs the undisputed merit of first recognizing the connection of the clinical picture with the anatomical condition, and of fully understanding the great importance of the former. After his first report in the Danish language (1868) he studied the affection further and in 1873 published a description of the disease in a German periodical, and twenty years later (1893) he enriched the pathology of the affection by valuable communications. Not only did he discover that the adenoid pharyngeal vegetations were an exceedingly important affection of infancy, but he energetically endeavored to increase our knowledge of the con-

dition. The service which Meyer has rendered toward the bodily and mental welfare of our children is extraordinary, and the erection of a monument in Copenhagen by his compatriots since his recent death has proved that their esteem for a promoter of the health of the human race is just as great as is that for the victorious generals and successful statesmen in other countries.

What has subsequently been written upon adenoid vegetations is legion. An affection that is so enormously frequent, that presents such readily recognized symptoms, that offers such a beneficial object of professional activity, must naturally engage the interest of the medical world in a high degree and offer rich stimulation to supplementary clinical knowledge, to pathologic study, and to operative procedures. To recount this vast amount of literature is impossible and superfluous. Only the most important points will be mentioned, which are sufficient to give a clear picture of the disease according to our present knowledge, and to show in how many directions Meyer's creative thought has had a fructifying effect upon medical investigation.

For a better understanding of the symptomatology we shall begin with the *anatomical basis of the affection*.

The pharynx, as is well known, is rich in lymphoid tissue which is not exclusively localized to the tonsils, the base of the tongue, and the pharyngeal wall, but is also very profuse in the pharyngonasal cavity. This tissue is found not only at the tubal openings and in the mucous membrane but it also accumulates, particularly at the roof of the nose, into a semi-globular mass which is designated the *pharyngeal tonsil* or *Luschka's tonsil*. When proliferation of this lymphatic tissue occurs the pharyngeal tonsil, as a rule, is primarily affected, but the remainder of the lymph-tissue of the nasopharynx also enlarges (Hopmann, B. Fraenkel, and others), as is usually the case with the other structures of the lymphatic ring which participates in this hypertrophy, especially the tonsils.

If, as is frequently done, the disease is designated merely an "enlargement" or "proliferation of the pharyngeal tonsil," the extent of the affection anatomically is not exposed, but that change is indicated which principally produces the clinical phenomena and the relief of which is sufficient to bring about recovery. From this description it is evident that in adenoid vegetations we are not dealing with new structures but that it is chiefly the proliferation of a physiologic tissue and the tendency of the same to inflammatory processes which produces the morbid phenomena. The conception of an "enlargement" of the pharyngeal tonsil is naturally not sharply defined. If the proliferation is so extensive that the entire pharyngonasal cavity is filled, that perhaps the lower pole of the tumor attains the height of the palate, there can be no doubt of its pathologic importance. On the other hand, with slight enlargement, the opinions regarding the importance of the changes may be divided; very often from the clinical symptoms only will we be able to decide whether the pharyngeal tonsil is pathologically enlarged and must be removed, or whether it is of normal size. The pathologic significance, therefore, lies not in the growth itself, which does not require extirpation as does a growing fibroma or sarcoma,



but in the relation of the same to its surroundings, in filling the nasopharyngeal space; a plug of cotton embedded in this region would produce similar symptoms. It is true, not merely the symptoms of occlusion of the nose occur, but also a stasis of secretion and irritative phenomena of the mucous membrane of the nasopharynx, and with these secondary symptoms which are so intimately associated with the adenoid vegetations that they should be included among the essential factors of the same.

Enlargements of the adenoid nasopharyngeal tissue is a *widely distributed and exceedingly common affection*. W. Meyer conducted ethnographic studies relative to this point and made the surprising discovery that adenoid growths are present in Indians, Chinese, and Greenlanders likewise as in Europeans and in the mixed races of North and South America and India. It is generally assumed that the disease is more prevalent in cold, damp regions than in climates which are warm and dry; Körner, for example, gives higher figures for the Baltic Sea-coast than for the interior country.

In regard to the *frequency* of adenoid vegetations there are contradictory reports. Meyer estimated 1 per cent. of such cases in school-children—a figure which, in a previously unknown disease, must have seemed high. Kafemann found the affection among school-children in 7.8 per cent. of the boys and 10 per cent. of the girls. Schnuckmann demonstrated adenoid vegetations in 30 per cent. of the pupils, and Felix's figures range between 28.52 per cent. and 35.10 per cent. Körner calculates the number of cases in children living along the sea-coast as 36 per cent., and in 26 to 28 per cent. of those residing inland. The specialist in diseases of children will be inclined to agree *with the higher rather than with the lower estimates* as these statistics were obtained from school-children, while experience demonstrates that adenoid vegetations are present even in infancy and that with advancing age the growth has a tendency to involution rather than to enlargement. If these statistics had been extended to smaller children very high percentages would have been obtained. Compilations regarding the prevalence of adenoid vegetations between infancy and puberty would be of great value, as they would prove, as experience appears to indicate, that there is first a rise and then a decline in the frequency of the pathologic picture produced by adenoid growths.

*Is there a congenital enlargement of the pharyngeal tonsil?* This is a debated question, although there is much to favor the affirmative. In nurslings we find symptoms which indicate adenoid vegetations, and it is often learned that one of the parents suffered from like symptoms during childhood. A familial tendency to adenoids is not rare, and here we occasionally observe that only those children are affected who resemble the parent who had suffered from the same condition. In ancestral picture galleries the typical facial expression of marked adenoids may be followed from generation to generation. In favor of congenital adenoids is the association of other anomalies in the formation of the face (see below) which can scarcely be regarded as mere sequelæ of obstructed nasal respiration. For these reasons we must coincide with those authors (B. Fraenkel, Semon, Lublinski, and others) who regard adenoid vege-

tations, often at least, as a congenital anomaly, and therefore we are not concerned with a "proliferation" but with a "hyperplasia" of the pharyngeal tonsil. Nevertheless, it is unquestioned that chronic coryza, as well as infectious diseases which run their course with special implication of the upper air passages, have a deleterious influence upon the lymphoid tissue of the nasopharynx, in that they first give rise to swelling, or, if the pharyngeal tonsil is already enlarged, chronic inflammatory processes arise and thus stimulate hypertrophy. Here probably similar conditions are operative as are readily apparent in the faucial tonsil.

**Symptomatology.**—In the clinical picture of adenoid vegetations, there are numerous characteristic factors which, singly or conjointly, at first glance often raise a justified assumption of the existence of an enlarged pharyngeal tonsil.

Only in *infancy* can there be errors in diagnosis; then the symptoms are less exact and examination by means of the finger or the rhinoscope is difficult to accomplish. The frequent recurrence of coryza with a tendency to chronicity, snoring, the early appearance of aural catarrh, as well as difficulty in nursing, are always suspicious symptoms which, often from the first months of life, permit a tentative diagnosis of adenoid vegetations. In such infants the characteristic phenomena often develop to such an extent that extirpation of the growth becomes necessary as early as the sixth or ninth month of age. We are less inclined to associate *laryngospasm* or "*suffocation*" of children, that is, the sudden arrest of respiration, with proliferation of the pharyngeal tonsil. The nature and course of these attacks, their frequent combination with rickets, the often prompt effect of phosphorus treatment, all justify in laryngospasm the assumption of an acute, nervous, pathologic condition rather than of an affection due to a permanent anatomical change. Even in those cases in which the expiratory form of laryngospasm, the suffocation above-mentioned, exists as a permanent symptom, lasting until the second or third year of life, the extirpation of such adenoids as may be present is by no means followed by that prompt relief which should be expected with an existing relationship.

*In older children the symptoms of the affection are unmistakable.*

According to the degree of enlargement of the pharyngeal tonsil the *nasal respiration is partially or wholly inhibited*. This stenosis of the nasopharynx almost exclusively forces the *respiration through the mouth*. On account of the constantly open mouth such children are in frequent dispute with their associates, who regard the symptom as a bad habit and constantly remind them to close the mouth. Smaller children, who have not been weaned from the breast or bottle, take their food in brief draughts and become very restless. Many seeming faults of the wet-nurse, particularly an insufficient quantity of milk at each feeding, may perhaps be referred to adenoid vegetations. Larger children eat hastily in a constant endeavor to free the mouth, the consequences of which are incomplete mastication and the swallowing of large particles of food. There is sometimes shortness of breath in speaking, which, although less prominent than the disturbances of speech yet to be mentioned, is nevertheless disagreeable. With severe nasal occlusion this dyspnea becomes very conspic-



uous when ascending steps and in prolonged walking and may simulate a direct symptom of disease; thus I know of a boy who was taken away from school on account of a "cardiac defect," in whom nothing was found but extensive adenoid vegetations. This inhibited nasal respiration may in mild cases be only slightly prominent and very frequently is transitory. When adenoid proliferations which hitherto have not seriously interfered with nasal respiration, become enlarged from coryza or cause the retention of secretions, the clinical picture of nasal stenosis arises and may create a quite serious disturbance.

In addition, the *enlarged pharyngeal tonsil may itself be the seat of a catarrhal inflammation of the mucous membrane*, which, as was mentioned previously, is revealed by high fever of several days' duration with secondary enlargement of the lymph glands of the neck, or by a chronic catarrh. The latter condition chiefly involves the mucous membrane of the pharynx, producing an irritation which results in a constant rasping, cough and expectoration. This is often thought to be a naughty trait, for which the child is punished. Inspection of the pharynx in such cases will show the posterior pharyngeal wall to be reddened and frequently granulated (*pharyngitis granulosa*), with a trickling mucopurulent secretion which readily accounts for the "naughtiness" and "hysterical cough."

Besides this chronic irritative condition, in which the pharyngeal organs may participate through adenoid vegetations, these growths are occasionally also the immediate cause of acute inflammation of the pharynx itself. Thus, in *relapsing tonsillitis*, vegetations in the nasopharynx should always be thought of as they retain the infectious secretion which from time to time reaches the tonsils. In these conditions experience has shown that tonsillotomy alone does not always relieve, and that only the removal of the adenoids will prevent a recurrence of these inflammations.

The *internal portions of the nose* may be involved in a similar manner. By propagation of the catarrh from the pharynx to the nose swelling and redness of its mucous membrane occurs, especially upon the erectile tissue of the turbinated bones, giving rise to that condition designated by rhinologists *rhinitis hypertrophica*. The lower turbinated bone may decidedly occlude nasal communication so that the inspiration of air is interrupted within the nose before the obstruction in the nasopharynx is reached. This combination of adenoid vegetations and hypertrophy of the turbinated bones is of highly practical interest, for, as Heermann correctly asserts, it may happen that not until extirpation of the adenoids and the continued existence of the affection will the greater obstruction in the nose be sought and found. Other diseases of the nose are familiar to the specialist as accompanying phenomena of adenoid proliferations, the symptoms of which are principally a muco-purulent nasal discharge and a *deficient sense of smell*.

The sequelæ of this nasal affection, which may readily be misunderstood, are *eczema, fissures, swelling of the upper lip*, which are so common with a profuse purulent flow from the nose. A diagnosis of scrofula is then apt to be



made and, based on this view, together with the frequent irritative condition of the respiratory organs and the enlargement of the lymph glands, next to be described, the assumption is raised that the adenoid proliferations are the expression of a scrofulous diathesis. That this view is not correct will be decided later.

In the description of acute inflammations of the nasopharynx attention was called to the importance of the enlargement of the superficial cervical glands, including those of the nape of the neck, in the diagnosis of such conditions. It is evident that from the acute swelling in chronic irritative processes of this region chronic intumescent lymph-glands may develop, and as a matter of fact chains of *pearl-shaped enlarged lymph-nodules behind the sternocleidomastoid (glandulae cervicales laterales)*, associated with catarrhal irritation, are the *most common accompanying symptoms* of such adenoid vegetations. As, however, the nose, the pharynx, and the skin of the lips may be the site of long-continued inflammatory phenomena, all of the glands may swell whose root area corresponds to these regions, and thus a well-simulated scrofulous clinical picture is produced. Even after extirpation of the adenoids and under favorable circumstances some time is required for the glands to return to their normal size.

The *ear* suffers to a great extent on account of the vicinity of adenoid vegetations. Not only is the opening of the Eustachian tube permanently or transitorily occluded by the proliferations and the entrance of air into the ear inhibited, but catarrhal processes from the pharyngonasal cavity are readily conducted to the tympanum. This is a serious consequence of adenoid vegetations, the full importance of which can be best realized from an expression of Mackenzie's that the discovery of adenoid growths has saved at least 100,000 persons from deafness. As a matter of fact, disturbances of hearing are present, according to Abeles, in 57 per cent., according to Halbeis in 74 per cent. (Hartmann and W. Meyer) of patients with adenoid vegetations. The oft-recurring acute diseases of the ear in the course of coryza must particularly awaken the suspicion of adenoid vegetations, which, on account of the severity of the possible sequels, require a radical treatment more than any other symptom of the affection. In scarlatina these proliferations may be of ominous import, for at least they favor the appearance and prolong the cure of an aural process.

The *larynx* and *bronchi* are among the organs which may be irritated through the presence of an enlarged pharyngeal tonsil. An important factor in the development of a laryngeal catarrh is the propagation of chronic catarrh from the nasopharynx, due to the deficient nasal breathing, whereby a valuable protective measure for pulmonary respiration is lost. As the air passes through the nose it is cleansed of the grosser admixtures, is warmed, and finally is saturated with moisture (Bloch and others), all of which processes are abolished when nasal respiration is inhibited. The result of these conditions when adenoid vegetations are present is the frequent occurrence of *pseudo-croup*, as well as the ready appearance of febrile bronchial catarrhs. I know of a boy

aged four years who, every few weeks for two winters, had an acute bronchial catarrh, almost asthmatic in character, associated with high fever, which was immediately and permanently relieved after nasopharyngeal proliferations were removed. In children who "cough the entire winter," who have frequent attacks of bronchitis and sometimes a circumscribed pneumonia, it is well to inform ourselves in regard to the nasal condition before speaking of "affected lungs" or "beginning catarrh of the apex." Blood-tinged nasal mucus not infrequently may simulate pneumonic sputum.

The symptoms of adenoid proliferation which have been described are in direct relation with the anatomical changes in the nasopharynx and its surroundings.

We shall now pass to the *other accompanying symptoms* of the affection which are of scarcely less importance for the development and health of the child.

A *change in the facial expression* under some circumstances is a very striking feature of the disease. With a normally closed mouth the tongue forms a sort of suction valve which tends to uphold the lower jaw. When nasal respiration is inhibited and the mouth remains open this suction power is absent and the lower jaw drops mechanically. The face is then elongated, the nasolabial folds obliterated, the expression is less animated; briefly, an appearance anything but intelligent is produced which, in combination with the mental condition of these children, soon to be described, appears to justify a low estimate of their intellect. This stupid physiognomy is very characteristic and when well-marked at once reveals to the expert the presence of adenoid vegetations; even in antique statuary and in old portraits this sign of adenoid vegetations has been observed.

A peculiar and as yet unexplained sequel of marked adenoid vegetations is *deformity of the upper jaw*. The bone is narrowed, higher, and forms an acute angle anteriorly, causing displacement of the teeth. How this malformation of the jaw arises is very questionable. Some authors regard it as a sign of degeneration, which naturally would also favor a congenital existence of adenoids. Other investigators regard it as the mechanical effect of constant mouth breathing upon the growing bone (Bloch, B. Fraenkel, etc.) and still others see therein a compensation for the retarded nasal development (Körner, Abeles).

That *disturbances in the growth of the nose* also occur from adenoid vegetations has been maintained by various authors.

*Flattening of the thorax and deviation of the vertebral column*, which has been described by a number of writers as a consequence of adenoid vegetations, is just as unintelligible and uncertain, but is probably the connecting link of an existing rachitis. Ziem has attempted to produce this deformity experimentally by sewing together one of the nasal openings in young cats, which was followed by scoliosis. Naturally these deformities of the thorax add to the disturbed respiration of children who suffer from an enlarged pharyngeal tonsil.

*Change in the speech* is a further factor which at once denotes adenoid vegetations. It has already been mentioned that a certain haste in speaking and

a disinclination to talk are sequels of the dyspnea due to occluded nose. More important and characteristic is the speech itself. Such children are often very late in learning to talk; for a long time they mumble their words, the nasal as well as the guttural sounds being indistinct. The principal feature, however, is the lack of timbre in the speech, which is brought about by the resonance in the nasopharyngeal cavity, so that a monotonous drone results ("dead speech," W. Meyer). *Stuttering* also is associated with adenoid vegetations in some cases. These disturbances of speech are not only significant factors in the recognition of the affection but are also an important indication for the specialist in speech disturbances, who very often must begin the treatment for delayed speech, stammering, or stuttering by removal of adenoid growths.

Whether the *headache* which is so common in children with adenoid vegetations is to be referred to an irritation of the nerve trunks adjacent to the nose, to affections of the auxiliary cavities, which in the child are but slightly developed, to swelling of the nose, to venous stasis of the vessels of the head, or to overloading of the blood with carbonic acid, as is maintained by many authors, cannot be answered. It is certain, however, that this disagreeable affection sometimes disappears rapidly after extirpation of the growths.

The headache or sensation of pressure upon the forehead, the difficult respiration, the disagreeable sensations in the nose, the dulness of hearing, and perhaps also a hindrance in the off-flow of intracranial lymph (Guye) combine to produce a *peculiar mental condition* in children with extensive adenoid vegetations which Guye has designated *aprosexia nasalis*, and to which Bresgen has drawn attention in the condition of school-children. Children of this sort seem languid and tired, they do not show the cheerfulness common to their age, are absent-minded, stare vacantly in school, and arouse as if from a dream when they are suddenly called. If these children have the unintelligent expression described above, which is so common with adenoid proliferations, the parents, and particularly the teachers, regard them as weak-minded and an inevitable encumbrance of the school, to whom attempts at instruction are useless. Or, as is not infrequently the case, the teacher discovers that when the child is forced to momentarily concentrate his attention he is not so stupid and ignorant as would appear, which forces the conclusion that he is purposefully inattentive and indolent; therefore punishment is instituted where treatment should be applied. How much this injustice reacts upon the child who realizes his shortcomings, how much family suffering, how many unsuitable occupations and miserable social positions are the outcome of these unrecognized consequences of adenoid proliferations is incalculable, and places in its full light the beneficent act of Meyer who has taught that all of these misfortunes might often be avoided by a slight operation. In the last few years school physicians and teachers have begun to devote some attention to these conditions and interesting statistics have been published. Thus, in 62 backward students Permewan found about 45 per cent., in 52 who were moderately intelligent about 30 per cent., and in 89 bright pupils about 21 per cent. with adenoid vegetations. Felix found among 1,038 bright and moderately bright



students of the middle classes 28.52 per cent., and in backward pupils about 35.10 per cent. with adenoid proliferations (quoted from Kotelmann).

When we consider the multifarious and serious symptoms of disease which may be definitely referred to proliferations of the pharyngeal tonsil it is not surprising that adenoids are held responsible for a number of other affections of infancy and have become a general source of disease in pediatrics.

From the *loss of appetite*, the *vomiting*, and the *deficient bodily development* of such children an explanation of this kind is comprehensible and justified, as every chronic pathologic process in the nose, pharynx, and ear is liable to interfere with the intake of food.

*Disturbances of sleep* may also find their explanation in adenoid proliferations. The hindrance of free respiration causes a frequent awakening, and in the morning the child arises pale and tired. In this sense Rey's hypothesis that *night-terrors* often depend upon carbonic acid intoxication resulting from the exclusion of air by adenoids is plausible, and in severe cases justifies the removal of nasopharyngeal proliferations.

More doubtful is the connection of adenoid vegetations with *enuresis nocturna*, notwithstanding the fact that many authorities maintain such a relation (Ziem, Schmaltz, Körner, Grönbech, and others). The assumed pathologic process is somewhat artificial and on account of the great psychical influence upon the affection the result of adenotomy is by no means decisive.

A relation of adenoid proliferations to *asthma* is favored, among others, by Hack and Chiari, to *salivation* by Conetoux, Haug and Rey; the possibility of a secondary *meningitis* is maintained by Hopmann and Roth, and an association with *ocular symptoms* (conjunctivitis) by Snellen and Guye. Attempts to cure *chorea*, *epilepsy*, and every possible form of *idiocy* by way of the nose are extremes readily calculated to reduce the great importance of adenoid nasal proliferations in the pathology of infancy and must be depreciated by physicians and patients.

A few remarks must be devoted to the relation of adenoid vegetations to *scrofula* and *tuberculosis*.

Meyer has considered the question of the *scrofulous foundation* of adenoid vegetations but is not very enthusiastic concerning a relationship. Some authors have maintained such a connection (Trautmann, Bresgen, Semon, etc.) but have met with great opposition from others (B. Fraenkel, v. Lange, Haug, Ziem). At this time the majority of pediatricists incline to the latter view; the experience of private practice in particular shows that the "scrofulous habitus" is rarely associated with adenoid proliferations, and that even in those cases in which suppuration of the nose and enlargement of the glands may perhaps, by a liberal diagnostic standard, be referred to a "beginning scrofula" such symptoms disappear after removal of the adenoids. By a mindfulness of these facts we may add to our exact knowledge of the symptoms and sequels of retronasal proliferations a valuable aid in disintegrating the by no means secure structure of scrofula. It would be in the interest of pediatric lucidity if the collective conception of scrofula were to disappear, resolving itself into

its essential parts. Besides tuberculosis, syphilis, pediculosis, etc., adenoid proliferations in the nasopharyngeal space occupy a not small portion of the realm of scrofula. This, however, does not deny that changes in the mucous membrane due to adenoid growths may continue to exist after removal of the proliferation, and that then, as we have demanded for scrofula, a predisposition arises in the body and not an external cause for the continuance of diseases limited to the regions of the mucous membrane and lymph glands. In this sense adenoid proliferations might be the starting-point of scrofulosis.

*Tuberculosis* also may find its port of entrance in adenoid vegetations. This view is favored not only by clinical experience, the interpretation of which may, however, be at fault, but by the anatomical findings, in that tuberculous nodules—i. e., caseation—have not infrequently been discovered in the extirpated pharyngeal tonsil (Brindel, Gottstein, Pluder-Fischer, Rethi, and others). To conclude from this that adenoid vegetations are the sequel of tuberculosis, or, for those who identify tuberculosis with scrofula, are due to the scrofulous constitution, would not be correct. It is much more reasonable to assume that tubercle bacilli which have entered with the inspired air are retained by the adenoid structures of the upper air passages and lead to local disease. This would be a new indication for the extirpation of vegetations, coinciding with the removal of a primary tuberculous focus.

**Diagnosis.**—The rich symptomatology furnishes enough supportive data to imply a diagnosis of adenoid vegetations from the clinical facts. For a positive diagnosis, however, a *local examination* is necessary, for which three methods are available: Anterior rhinoscopy, posterior rhinoscopy, and digital examination. Mere inspection of the pharynx will not reveal the lower walls of the proliferation except with very large tumors; much experience is required to determine whether the soft palate is forced forward, as is sometimes the case with extensive adenoids.

In regard to *anterior rhinoscopy* with the nasal speculum Chiari asserts: "After removal of the mucus, in a nasal passage that is at all dilated an elevation of the velum will be observed when the patient pronounces the letter *e*. It will be seen that the velum moves to the lower border of a light red swelling in the nasopharyngeal space; this mass has a straight or wavy margin and is itself raised by the velum so that the light reflex originating from its anterior surface distinctly shows the displacement." Although this mode of investigation is advantageous on account of its facility and the avoidance of pain, it is of little use in children's practice. On the one hand, expertness is necessary in recognizing the findings, which may not be familiar to every pediatricist, and on the other hand the nearness of the reflector frightens the child so that a quiet examination is difficult. This is all the more true of *posterior rhinoscopy*, the management of which requires great skill on the part of the physician as well as a certain amount of intelligence in the patient. The latter method, however, undoubtedly furnishes reliable results and permits the recognition of the adenoids, as swellings or growths, which are supplied for the most part with furrows and indentations.



In children's practice these methods can only be utilized in exceptional cases and in the hands of expert laryngologists. The most positive means of recognition of the adenoids and a method that is easily accomplished is *digital examination*.

For this it is necessary to extend an index finger to the height of the choana, behind the velum, with the head of the child inclined forward. The investigator first opens the mouth with a tongue depressor. The index finger (usually the right) is then inserted to the posterior wall of the pharynx, the tongue depressor is withdrawn, and the bent middle and terminal phalanges of the finger are next extended into the nasopharyngeal cavity. In this examination it is well for the physician to stand alongside the child, who is seated upon a low chair, so that he can steady the head with his free left arm; this is absolutely necessary when assistance is not at hand and the physician himself must fix the head of the child. The danger of biting is extremely slight when once the finger has reached the posterior pharyngeal wall; as a matter of precaution, however, the cheek may be depressed between the teeth by an assistant during the examination. This investigation must be very rapid on account of the decided gagging and resistant movements of the child and its fear of suffocation. The entire art of diagnosis with the palpating finger lies in the fact that in a few seconds a clear estimate of the condition may be obtained. The beginner often fails by allowing too much time for the child to offer resistance and by not forcing his finger high enough, so that the upper pole of the tonsil which narrows the passage is mistaken for adenoid proliferations of the nasopharynx. Lesions of the mucous membrane and slight hemorrhage are frequently produced during this examination but rapidly subside without treatment.

Another form of diagnostic exploration is occasionally advised. In doubtful cases, instead of the finger, a bent forceps is introduced from the mouth into the nasopharyngeal space and an attempt made to grasp any obstruction to its entrance. If lymphatic tissue is withdrawn adenoid proliferations are present; if not, they are absent. This procedure is not justifiable, as it eliminates the indications for operation, which for ethical reasons must be sharply defined, even in a relatively simple and beneficent surgical procedure.

If we are convinced by digital examination of the presence and consistence of adenoid masses in the nasopharynx, the diagnosis is positive. Only exceptionally are other tumors present in the nasopharynx, as fibroid nasal polyps, which are much coarser, or soft sarcomata, which bleed readily, or gummata, all of which must be considered in a *differentio-diagnostic* respect. Apart from the history and other signs of the disease, if the local condition is not sufficiently defined only a histologic examination of an extirpated particle and the further course will be decisive. Another important question is whether the mass in the nasopharyngeal space is the only source of the trouble; therefore the possibility of hypertrophy of the tonsil or of enlargement of the turbinated bones must always be considered.



**Prognosis.**—As regards life the prognosis is always favorable; only when severe complications (otitis, tonsillitis, meningitis, pneumonia, etc.) arise does it become serious. A spontaneous disappearance of the pharyngeal tonsil is very probable. Chiari states that the same conditions prevail with adenoids as with the palatine tonsil, which shows its greatest development between the fifth and eleventh years of life, and then gradually disappears. With the growth and enlargement of the nasopharynx the disturbances due to occlusion are sometimes overcome. The accompanying catarrhal condition declines gradually. We may then speak, in a clinical sense, of spontaneous recovery, even when the pharyngeal tonsil as such has not become smaller. Often enough, however, the symptoms of the disease continue even in adolescence, and we must then resort to operation.

**Treatment.**—Adenoid proliferations in the nasopharynx offer a fruitful field for professional activity, and the treatment of the affection has been developed in detail.

The only correct method of treatment, if this be at all necessary, is the surgical removal of the proliferations. Only with children and parents who fear the knife and with direct contraindications to an operation may applications be employed. This method is accomplished either with a fine probe covered with cotton, which is passed through the nasal cavity or, better, with a bent probe by way of the mouth. A 5-10 per cent. solution of nitrate of silver is employed. Great success is not to be expected from this treatment; at best, only the accompanying catarrh is improved. Treatment of this kind, however, is rarely completed; the patients object to the unpleasant method and declare to the physician that the condition has decidedly improved.

*When do adenoid vegetations in the nasopharynx require operation?* To this there can be only one answer: When they give rise to distinct, characteristic symptoms. The mere presence of an enlarged pharyngeal tonsil, without symptoms of disease, is just as little an indication for operation as the existence of indefinite factors which do not belong to the clinical picture of adenoid vegetations. It is true, experience shows that the indications for operation vary widely. In contrast with those physicians who are timid with the knife, who are only too readily influenced by the unnecessary fear of the parents, there are many others who regard every pharyngeal tonsil as the starting-point of complications and look upon a slight coryza as a signal for operation. If, in deciding upon operation, we are not guided by subjective impressions, which will not be undervalued by the experienced physician, but are governed by definite factors, then the relapsing otitis and a tendency to pseudo-croup or to febrile bronchitis in small children, and in older children difficulties in speech and aprosexia, are to be regarded as positive indications for operation. The following are contraindications for surgical intervention: Hemophilia and other diseases of the blood, acute febrile conditions, tonsillitis, and the epidemic appearance of diphtheria and scarlatina in the vicinity of the child. To these Abeles has added valvular disease and other affections which are accompanied by stasis in the course of the superior vena cava, epilepsy, severe hysteria, and

the presence of an intense ciliary injection of the eye. Neither infancy nor anemia are contraindications for operation.

For the *operation* a cutting instrument is introduced, in the same manner as the finger in digital examination, to clear the nasopharynx of adenoid tissue. To accomplish this with the finger-nail, as is occasionally suggested, is not advisable in the present era of the greatest possible cleanliness. The cutting-ring, to be adjusted to the finger of the operator, which was at one time advised (Justi, Bezold, and others) is not practical for the reason that the crowding finger interferes with a thorough removal of the proliferations. At present the *curette* and *forceps* are employed and many rhinologists subsequently use a *cold snare*.

The *curettes* which are in use are those of Gottstein, v. Lange, Hartmann, Gruber, Politzer and Fein, and others, all of which are composed of an egg-shaped, continuous ring with an inner cutting edge, arranged at a right angle upon a firm handle. The numerous modifications of the original Gottstein knife differ principally in the size and shape of the ring and its curvature, and in the position of the cutting surface. A very practical device is that of Fein, the handle of which is curved so that the field of operation is not obscured by the hand.

The *choana forceps* for the removal of adenoid vegetations (Catti, Jurasz, Schütz, etc.) has upward-curving branches, the ends of which terminate in a sharp spoon or edged rings. By this instrument the proliferations are cut and pinched off. At the present time these forceps are utilized for the removal of adhesions remaining after Gottstein's method has been employed, rather than for the actual operation.

The *steel snare* (Blake, Chiari, etc.) is introduced either through the nose or by the oral cavity. Both methods of application have their adherents. Chiari favors the nasal employment of the snare, Stoerk and others the retro-nasal removal. While the results of the snare are satisfactory in the hands of an expert and with quiet patients who are cocainized, the method is difficult in children's practice on account of the necessity of repeated introduction of the instrument and because of the employment of large doses of cocain, which is especially necessary with operation through the nose. Some operators use the snare after Gottstein's knife has been employed, as by this means the vegetations remaining in the pharyngeal vault are readily grasped and removed.

A greatly discussed question is whether *anesthesia* is advisable for the removal of adenoid vegetations. Some laryngologists resort to it invariably to obviate the possibility of resistance from the child and to insure a quiet and thorough operation. Others object to the measure because the shock of narcosis in children is usually very alarming, the expectoration of blood and particles of tissue is prevented, and finally because the operation may be done rapidly and easily without anesthesia and thus eliminate the complications of narcosis. If anesthesia is employed at all it should not be so profound that the reflexes are abolished; a mild etherization, such as is often employed in minor surgery, appears to be the most suitable for this purpose.



The *method of operation* is as follows: The child is held by an assistant, with the feet encased between his knees and one arm surrounding the body and fixing the hands. Small children are swathed, mummy-like, in a sheet which is fastened with safety-pins. With the other arm the assistant holds the head of the child firmly toward him. Two people are often necessary for this purpose. The operator sits opposite the child, opens the mouth with a tongue depressor, introduces the instrument into the pharynx, and thence into the nasopharynx. If, as is usual, the Gottstein instrument is employed, strong scraping movements from left to right and from above downward are necessary, during which the experienced operator can determine whether he is still working upon soft tissue within the nasopharynx. When the instrument is withdrawn the head must be inclined forward so as to permit a free path for the profusely flowing blood. The adenoid tissue which has been scraped away may adhere to the knife or be expectorated with the blood, but very often the masses are swallowed and digested, sometimes vomited. The operation lasts a fractional part of a minute. The hemorrhage soon ceases, but before the patient is released the operator should convince himself by inspection of the pharynx and digital examination that no shreds of the proliferation are hanging toward the pharynx and that the adenoids have been completely removed. Some rhinologists always introduce the cold snare into the nasopharynx after the operation, either by way of the nose or the pharynx, to remove remaining particles from the roof of the nose. Loose shreds may readily be removed by the Jurasz forceps, which is sometimes preferred for the operation. After the operation is completed the patient should remain under the eyes of the operator from thirty minutes to an hour, or until the hemorrhage has entirely ceased.

The *after-treatment* is exceedingly simple. If local measures are necessary daily insufflations of dermatol, sodium sozoiodol, etc., through the nose may be employed. Small children should be kept in bed for a day or two and older ones for at least four or five days in a room in which there is plenty of ventilation but no dust. The food in the first days is to be fluid or soft. Ice-cream after the operation is often soothing and quickly relieves the unpleasant sensations. The process of healing is smooth and uninterrupted; only in rare cases are there local inflammatory phenomena, described above.

*Relapses*,<sup>1</sup> that is, the *necessity for subsequent operation*, may be noted even when the primary operation was thoroughly done. This is particularly true of the operations upon infants—a circumstance which should be considered with the indications for operation at that period of life. Three per cent. of relapses, estimated by N. Schmidt, is probably too low, as patients often seek a different nose specialist for relapses. Of greater value would be the reports of family

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<sup>1</sup> Many "relapses" in the shape of adenoids or of chronic catarrh after the operation result from the fact that the operators, mainly those who specialize, are satisfied with the "operation." A chronic nasal catarrh, a sore surface left behind after the scraping with Gottstein, and the tendency to adenoids, require two daily warm saline irrigations of the nares. This simple manipulation renders many an operation superfluous.—EDITOR.



physicians, who are in a better position to know whether symptoms of nasal stenosis reappear after removal of the proliferations.

The *result* in by far the majority of cases is convincing and permanent. Improvement in speech, the possibility of blowing the nose, the pleasant sensation of free nasal respiration, quiet sleep without snoring and with closed mouth, are sequels which may often be recognized within the first twenty-four hours after the operation. No fear need be aroused if, two or three days later, there is an apparent aggravation, brought about by a reactive swelling of the surface of the wound. This soon disappears, giving place to permanent health. The rapid mental and bodily development of a child who had previously suffered from the depressing symptoms of adenoid proliferation is often surprising.

## DISEASES OF THE PHARYNX

*Diseases of the pharynx in childhood* do not come within the scope of this article as they do not differ from similar conditions in adults and have been described at another place in connection with tonsillitis.<sup>1</sup>

In children *acute catarrhal pharyngitis* occurs as a substantive disease or in association with inflammation of the palatine arches, the tonsils, the nose, and the nasopharynx; *chronic inflammations* of the mucous membrane of the pharynx are observed principally in diseases of the nose and in adenoid vegetations. Not rarely do we meet with typical forms of *pharyngitis granulosa*, and *phlegmonous* varieties are found which are mostly associated with a corresponding disease of the tonsils and the palatine arches. Finally, the various pharyngeal affections appearing in the infectious diseases, particularly in diphtheria, measles, and scarlatina, must be mentioned which, as a rule, are very characteristic and of decided diagnostic importance.

All of these conditions are common in children's practice, certain of them being more frequent than in adults, such as inflammation of the tonsils and the pharyngeal affections in infectious diseases, and in infancy particularly they are often succeeded by lymph-gland enlargement and disease of the ear. For the pediatricist there is an especially important relation between relapsing tonsillitis and acute endocarditis, and between articular rheumatism and chorea.

A more comprehensive description will be devoted to a form of disease which is peculiar to children, and has not been given the importance it deserves, namely

### ACUTE LYMPHADENITIS RETROPHARYNGEALIS

By *lymphadenitis retropharyngealis* we understand an acute febrile enlargement of those lymph nodules which are situated upon both sides of the median line of the posterior pharyngeal wall. Bokai, senior, in his pioneer report upon retropharyngeal abscess (1857) called attention to the non-suppara-

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<sup>1</sup> See volume on "Infectious Diseases."

tive inflammation of these lymph-glands, which he sharply separated from acute pharyngitis, but the interest of physicians has been more especially devoted to the suppurative varieties of this glandular inflammation and the knowledge of simple lymphadenitis, notwithstanding Baginsky, Neumann, and others, is very slight.

The affection usually begins with the symptoms of an acute pharyngeal inflammation, from which, under peculiar bacterial conditions, it probably always originates. The fever is not extreme—between  $38^{\circ}$  and  $39^{\circ}$  C. ( $100.4^{\circ}$ - $102.3^{\circ}$  F.)—sometimes intermittent in character, perhaps with remissions of several days. The constitutional condition of the child, however, is as a rule decidedly changed. At first there is difficulty in deglutition and headache. The tongue is heavily coated and the appetite lost. With small children proffered fluids may be quickly grasped but after a few attempts to swallow the food is resentfully rejected. Inspection of the throat reveals redness of the pharynx; examination with the finger at first shows merely a loosening, unevenness, and swelling of the pharyngeal mucous membrane, which readily bleeds upon irritation with the finger-nail. After one or two days, and often from the onset of the disease, the palpating finger discovers, laterally from the median line, a soft, elastic, non-fluctuating, elongated, apparently painful enlargement on the posterior wall of the pharynx, at the height of the palate or usually somewhat deeper. This enlargement corresponds to a pharyngeal lymph-gland, with which we have become familiar through the investigations of Most. Therefore, we are apparently concerned with an inflammation of the profuse net of lymphatics situated upon the posterior wall of the pharynx, with consecutive swelling of the adjacent pharyngeal lymph-nodes. In this stage of the disease another symptom arises which is much more prominent than the transformation in the interior of the pharynx and to unobservant parents is sometimes the first indication of the trouble. This is a *moderate enlargement of the lymphatic glands at the angle of the jaw*, that is, on the anterior border or under the insertion of the sternocleidomastoid, belonging to the group of deep cervical glands.

This lymph-gland enlargement is a troublesome as well as conspicuous symptom. In the course of a few days, and sometimes within a few hours, these glands (one or two upon each side) enlarge to the size of a nut or even a small apple, causing a decided deformity. Sometimes the swelling is only unilateral, but as a rule it is unequally distributed to both sides.

The greater enlargement corresponds with the side of the lymph-gland inflammation in the pharynx. The localization of the tumor under the mastoid process, the commonly severe pain upon pressure, the doughy infiltration of the surrounding area, which at first makes a sharp limitation of the gland impossible, closely resemble mumps, for which the affection is often mistaken. A slanting position of the head and decided resistance of that member to passive movement are the ordinary consequences of this painful glandular enlargement.

The swollen region at first is usually of a soft, elastic consistence, apparently due to edema of the parts surrounding the gland. When the edema dis-

appears the gland is recognized as a hard, coarse tumor. Fluctuation and supuration are rare.

The disease may remain in this stage for eight to fourteen days. The restlessness of the child, caused by the great swelling of the external lymph glands, the inadequate nourishment in consequence of inflammation of the retropharyngeal gland, the fever, the constant disturbance of sleep, stamp the condition as serious, but rarely is it dangerous, although the patient is considerably emaciated. After watching this condition for several days the physician will often conclude to incise the tumor in the posterior pharyngeal wall with the expectation of finding a suppurative focus, not perceptible by palpation, or for the purpose of relieving tension. Upon decline of the fever recovery ensues, but the glands under the insertion of the sternocleidomastoid usually remain enlarged for some time. In other cases a gradual abscess formation results from this retropharyngeal adenitis which exhibits the typical symptoms of retropharyngeal abscess and heals rapidly after incision.

In one of my cases fourteen days elapsed before the lymphadenitis proceeded to suppurate.

The above-described course of acute retropharyngeal adenitis was observed by me in 6 cases, the ages of the patients ranging from nine months to five and a half years. Two of these children were related and were affected in rapid succession, not by direct contact but through a third person who suffered from a mild tonsillitis. A clearly epidemic occurrence of the malady has been reported several times (Bokai, etc.).

**Prognosis.**—The prognosis of simple acute retropharyngeal lymph-adenitis is good, although the process of healing is sometimes greatly protracted and the child is much emaciated. In small children the difficulty in taking nourishment and the fever may make the condition menacing. There is also a possibility of mechanical hindrance of respiration and of edema of the glottis; the danger is then as great as in retropharyngeal abscess—perhaps more so, because the relief of the condition is not so simple. These symptoms, however, are more rare in acute lymphadenitis than in abscess.

**Treatment.**—The treatment is purely symptomatic and consists of vapor packs with water or dilute aluminum acetate, the external application of iodine or ichthyol in salve or vasogen, together with gargles, applications to the pharynx, etc.

### RETROPHARYNGEAL ABSCESS

In intimate relation with these enlargements of the lymphatic glands is *retropharyngeal abscess*. Even in his early report of this disease Bokai, senior, sharply differentiated it from the varieties of acute pharyngitis, declaring it to be a suppurative inflammation of the glands behind the pharynx. The anatomical basis of this clinical observation was proven later by Most and others who demonstrated the presence of lymph glands in the posterior pharyngeal wall.

Since the first publication regarding retropharyngeal abscess by Bokai,



senior (1857), cases have been reported by a number of other authors (Alexy, Henoch, Schmitz, Bokai, junior, Temoin, Oppenheimer, Dollinger, Neumann, Koplik, Sokoloff, Burekhardt, and others) and at the present time we possess a very comprehensive knowledge of the disease.

**Symptoms.**—In contrast to non-suppurative lymphadenitis *retropharyngeal abscess* principally occurs in the first year of life. Henoch classes the affection in his text-book among the diseases of nurslings. The prodromal symptoms and the fever are not severe and the local phenomena are usually the first noticeable indications of the affection. These consist of rejection of food, swelling of the glands, snoring, increasing difficulty in respiration. After a few attempts at the breast or bottle the child unwillingly lets go as deglutition causes pain and also inhibits respiration. In the words of the mother, breast-children are weaned of their own accord. The enlargement of the glands under the insertion of the sternocleidomastoid is usually not so great as in the non-suppurative lymphadenitis of the posterior pharyngeal wall, although it is rarely absent. Instead of the distressing unilateral position of the head there is often a forward inclination which is due not merely to the enlarged lymph-glands in the neck but also to the restriction of respiration by the abscess.

The latter is the most important and serious symptom of the disease.

The respiration at first has a peculiar snoring quality which is often assumed to result from coryza and is especially pronounced during sleep. This hindrance of respiration gradually becomes more prominent, the child sleeps poorly, awakens readily, and, as Henoch expresses it, gasps for air. A condition may now arise which is as distressing for the parents as it is serious for the child. Breathing even with the aid of the auxiliary muscles is difficult, the child is cyanotic, inclines the head forward, and energetically refuses food, as even a brief closure of the mouth during feeding gives rise to suffocation; briefly, there is the well-defined picture of respiratory stenosis which is only too often encountered in diphtheria. Even at this stage, unfortunately, the malady is often unrecognized and is mistaken for laryngeal croup. Bokai tells of a surgeon who was called in haste to perform a tracheotomy in a supposed case of diphtheria but was unable to save the child, who was in the agonal stage, by an immediate incision of a retropharyngeal abscess which he found instead of diphtheria. I also know of a child who, the day after I had incised an idiopathic retropharyngeal abscess, was admitted to a hospital for diphtheria, probably because the wound of the incision was coated and the still-existing dyspnea, in the absence of a clear history, resembled laryngeal diphtheria. In these cases, aside from the history, only the cough can be considered as proof, which lacks the barking, hoarse character of the laryngeal affection. Henoch, however, reports cases in which the nature of the cough was uncertain on account of a secondary laryngeal catarrh which rendered a correct diagnosis exceedingly difficult. Such examples make it evident that in all cases where the history is unknown a digital examination of the pharynx is necessary; in addition to retropharyngeal abscess the possibility of a foreign body requires this measure.

*Digital examination* in retropharyngeal abscess reveals a condition which is unmistakable. The posterior pharyngeal wall is felt to bulge markedly and a fluctuating tumor the size of a hazelnut or chestnut is readily palpable. This, like the non-suppurative adenitis, is situated laterally from the median line, but with a fully developed abscess which completely fills the palpable posterior pharyngeal wall its position is not easily defined and a day often elapses after the pus has been discharged before it is possible to determine from which side the abscess originated.

When the physician has decided that a retropharyngeal pus cavity is present *incision should not be delayed*. This operation is a vital necessity, for with delay either a serious asphyxia, previously described, may arise or spontaneous rupture of the abscess may permit pus to escape into the respiratory passages causing, sudden death or a secondary deglutition pneumonia. Hensch reports an instance of this kind: A physician who wished to demonstrate a case of retropharyngeal abscess upon the following day lost his patient during the night by an attack of suffocation. Even an incision made at the proper time is not without danger. During and after the operation pus may find its way to the respiratory passages, giving rise to dyspnea and later a deglutition pneumonia (Temoin). I myself remember a case in the clinic in which I opened an abscess without any special difficulty and after cleansing the wound and arresting the flow of blood sent the child home. About an hour later the mother returned with the report that the child had died suddenly on the way home.

The internal incision should never be undertaken without sufficient assistance. One person holds the child, closely enveloped in a sheet as for an intubation, while another fixes the head firmly and, immediately upon the signal of the operator, inclines the head forward so as to facilitate the flow of pus through the mouth. There has been much discussion concerning the instrument with which the incision should be made. Some authors utilize the fingernail, but this does not fulfill the modern requirements of cleanliness and the insufficient opening is very liable to close. Usually a pointed bistury is employed, the cutting edge being covered by adhesive plaster. Some authors have invented special instruments for this and similar operations but they are employed chiefly by specialists.

In my experience the best results have been obtained by a simple *drainage forceps*,<sup>1</sup> which may be sharpened at the end if desired. The advantages of the instrument, which is employed also by Huber, are that it has no cutting

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<sup>1</sup> The "simple drainage forceps" recommended by the author and Dr. Francis Huber, that distinguished all around doctor and teacher, may refuse now and then when the soft parts outside the abscess do not give way to a blunt instrument like a forceps. Then a strong probe, sharpened or not, or a director will pierce the parts, and may be utilized to enlarge the wound, mostly downwards.

The lateral operation, from outside, is not so dangerous as the author seems to suggest. No neighboring blood-vessels need be feared, for the incision is made outside and behind the cleidomastoid muscle, and the operation can be finished with blunt instruments.—EDITOR.

edge and therefore does not produce secondary injury, that after insertion into the pus focus the arms of the forceps may be opened to any desired extent, that every physician possesses the instrument, and that it is readily cleaned and is not spoiled by the operation.

*Evacuation of the abscess does not complete the duty of the operator.* In addition it is necessary to note that the pus has been completely discharged and that the bleeding has ceased. To determine the former the cavity may be gently stroked from below upward. When the discharge of pus is profuse the child should not be permitted to go home on account of the danger mentioned above. Washing of the field of operation through the nose or pharynx is not advisable as the dyspneic child is already much frightened and distressed and on account of the impossibility of expectoration there may be cough and perhaps aspiration of some of the cleansing fluid. Removal of the pus from the mouth will usually prove sufficient. As a rule the hemorrhage is not profuse, but if necessary compression may be practised with pellets of cotton. The abscess must be inspected again upon the following day and if it has refilled the operation must be repeated.

The dangers of pharyngeal evacuation of the pus have led to other operative proposals. Thus, it has been suggested to operate with the head hanging—a method which has proved of service in many operations upon the pharynx and mouth. This will not be the preferred operation in a very dyspneic child and also requires an additional assistant and a greater amount of skill than in the simple opening of the patient's mouth. Neither is the procedure without danger, as is shown by a case reported by Zadock. Another method, strongly advocated by Burckhardt, is the external opening of the abscess, especially when it begins to protrude. Undoubtedly this is the correct surgical treatment as it permits of antisepsis, which is impossible with the internal method. This operation can be performed only in a well-appointed surgical dispensary and is not devoid of danger on account of the vicinity of the great vessels and nerves. A general employment of this measure, therefore, would result in fewer operations for retropharyngeal abscess and would not accrue to the advantage of the patients. The rejection of this method by Bokai, junior, which is based chiefly upon the good results obtained in the Children's Hospital at Budapest by the pharyngeal operation is therefore justified.

That the result of the operation, in an overwhelming majority of the cases, is brilliant has already been stated, and few operations as simple have met with such great success. Bokai, junior, reports that among 138 cases operated upon under his direction 106 were subjected to the internal method, of which only three fatalities among the dispensary patients could be considered as due to the operation. It is true there are cases with and without operation in which the pus takes an abnormal course and gives rise to serious complications. Henoch reports a few instances in which external rupture occurred behind the pharynx and into the ear. Inversely, particularly in scarlatina, a phlegmonous inflammation of the connective tissue of the throat may rupture into the pharynx and lead to discharge of pus through the fauces.



From these casuistic considerations it will be observed that if idiopathic retropharyngeal abscess is recognized and treated early its course is usually without danger, but if neglected it may become an exceedingly serious malady.

The correct diagnosis and the proper treatment should become the common property of all physicians, just as is the performance of a life-saving tracheotomy. In any throat affection that is at all obscure a digital examination of the pharynx at least should be required as the failure to recognize this affection would then be almost impossible.

**Etiology.**—Thus far in our discussion of “idiopathic” retropharyngeal abscess we have not considered its etiology or its relation to other diseases. Regarding these questions there is some uncertainty.

The existence of a constitutional basis (scrofula, tuberculosis), or at least of a chronic local affection of the mucous membrane of the pharynx and nose, is assumed by some authors (Bokai, Baginsky, Walls), and denied by others (Schmitz, Henoeh, Stoos). Dollinger’s case, upon which an autopsy was held, occurred in a tuberculous individual but without any evidence of tuberculosis in the abscess itself. For the professional conception of retropharyngeal abscess it is sufficient to know that the malady is observed in children who neither were nor have become scrofulous or tuberculous. This, however, does not exclude the possibility of a predisposed condition of the mucous membrane.

*Bacteriologic* investigations regarding *idiopathic retropharyngeal abscess* have been reported by Koplik; in all cases this author found *streptococci*.

Retropharyngeal suppuration is sometimes associated with *influenza* (Fischer, Stoos). So long as this connection depends merely upon clinical evidence, not upon bacteriologic findings, the association must be regarded as doubtful. In the discussion of Pfeiffer’s glandular fever it was remarked how often obscure febrile conditions in children sail under the false colors of *influenza*.

In *scarlatina* retropharyngeal abscess is not rare. It is then a secondary condition of the phlegmonous pharyngeal process which may result in a collection of pus over the entire posterior pharyngeal wall. Cases of this kind are exceedingly severe and even incision of the abscess does not often give relief; sometimes an aggravation of the local condition is produced by necrotic destruction of the walls of the abscess.

*Spondylitic gravitation abscess* must also be mentioned. This occasionally forms upon the posterior wall of the pharynx, simulating a retropharyngeal abscess. A differential diagnosis of these from idiopathic retropharyngeal abscesses is considered theoretically more often than in practice; only exceptionally will the spondylitis fail to produce symptoms and the disturbances of the retropharyngeal tumor alone call attention to the gravitation abscess in this locality. However, in this secondary phenomenon of cervical spondylitis we are no longer concerned with a retropharyngeal abscess, in the strict sense of the term, but with a suppuration appearing upon the posterior pharyngeal wall which has originated at some remote point.

In conclusion, the possibility of a *traumatic origin* of retropharyngeal ab-

scess must be mentioned. Such a condition is favored if a hard substance has been swallowed or has lodged in the throat, producing a lesion of the pharyngeal wall and a secondary suppuration. Substances accidentally introduced into the mouth may give rise to the same consequences (particles of glass, splinters of wood, etc.).

From these considerations it is evident that in addition to the "idiopathic" retropharyngeal abscess of infancy the condition may arise secondarily in various diseases. In none of these cases is it possible to exclude streptococcus infection as the basis of the trouble, as is shown by the investigations of Koplik and the repeated experiences in scarlatina.

The knowledge of adenoid vegetations and of retropharyngeal abscess are acquirements of the last decades. We must be grateful to W. Meyer and Bokai, senior, for their explorations which have opened new paths for pediatric investigation as well as for the beneficial activity which they have furnished to the physician.

# MENINGITIS OF INFANCY, AND HYDROCEPHALUS

BY O. KOHTS, STRASBURG

## SIMPLE MENINGITIS

THE brain is enveloped by membranes, the dura mater, the arachnoid, and the pia mater. The dura mater covers the brain at predominant areas and particularly at the base, is firmly adherent to the bone, sends ramifications into the foramen cranii, and is continuous with the periosteum. In children this membrane is less adherent to the skull than in adults. The meningeal vessels are found in these fibrous integuments. The dura mater is comprised of an external and an internal layer. The inner layer sends branches to the various cavities of the brain.

The arachnoid also has two layers, one lying close to the inner surface of the dura mater and covered with a single epithelial layer, while the visceral surface comes in contact with the pia only at the prominent portions of the cerebral convolutions. Both layers unite at the nerve roots and sometimes accompany them for quite a distance. Between the layers there is but little serum. A narrow space between the pia mater and arachnoid contains the cerebrospinal fluid.

The pia mater is a very delicate tissue, rich in cells, which lies directly over the brain. It surrounds the cerebral convolutions and fossæ, permeates deeply, and forms the choroid plexus of the lateral and middle ventricles. Its inner layer is continuous with the greater number of vessels and also forms the sheath for each nerve. It is uncommonly luxuriant in blood and in nerve fibers.

The cerebral membranes continue to the spinal cord. The arachnoid is never alone implicated by inflammation, but is inseparably involved in inflammation of the dura (*pachymeningitis*) or of the pia (*leptomeningitis*).

The term "meningitis" designates an inflammation of the pia mater and of the visceral layer of the arachnoid. This inflammation arises from widely diverse causes of infection, and since the most varied pathogenic agents may produce the same clinical picture we are usually unable at the onset to form any conclusions regarding the nature of the infection. Exceptions to this are furnished by tuberculous meningitis and epidemic cerebrospinal meningitis, which in many cases present somewhat characteristic clinical phenomena.

Meningitic symptoms accompanied by fever are also noted in the course of



the most varied diseases, as pneumonia, enteric fever, influenza, dysentery and otitis media, as the result of intoxications, or as reflex phenomena.

The complete clinical picture of meningitis may be observed without the demonstration post mortem of any changes of the meninges. In the absence of manifest changes in the brain and of microorganisms in the cerebrospinal fluid and in the blood, the symptoms must be referred to toxins which produce vascular dilatation and cerebral circulatory disturbances.

A neuropathic predisposition, extraordinary mental development, the greater sensitiveness of the brain of children, are given as reasons for the more frequent occurrence of cerebral symptoms—the symptoms of meningitis—in youth. Exceptionally the signs of chronic meningitis are found at the autopsy, whereas during life there were no symptoms corresponding to that affection. Simple meningitis was first differentiated from tuberculous meningitis by Hopfengärtner (1802) and by Rilliet. The earliest investigations appertaining to the relation of meningitis and micrococci were made by Klebs. This author demonstrated post mortem the existence of diplococci in the exudate of an individual who had succumbed to the disease. Later Eberth and v. Leyden isolated cocci and diplococci, and finally Netter, Fraenkel, Saenger, Weichselbaum, Foa, Bordoni, Uffreduzzi, Jaccoud, Hutinel, Schäfer, and others, established the presence of the various micrococci which play a rôle in the non-tuberculous forms of meningitis.

Acute meningitis is more common in adults than in children. On the other hand, children are the more frequent victims of the tuberculous form. The disease usually attacks well-nourished individuals and is especially prevalent in the first two years of life and after the sixth or seventh year. Quite exceptionally it has been observed in the newborn (Billard and Guersant).

Micrococcus infection may be the result of a trauma or of the transmission of inflammation from the surroundings through the lymph channels, or by metastasis through the circulation. An especially predisposing factor of meningitis is furnished by inflammation of the nasal and orbital cavities, of the frontal sinus, and of the ears. Direct effect of the sun, injury of the skull, or even lesions of the cutaneous covering of the skull, may occasion meningitis, or it may originate from fractures or septic diseases of the cranium, osteomyelitis or septic conditions emanating from disease of the face and the skin, furunculosis, impetigo, anthrax, phlegmons, and extensive burns. Acute meningitis may occur either in the course or as the sequel of disease of the nose (coryza, ozena, tuberculosis, syphilis), or of the ear (suppurative otitis).

Secondary meningitis, due to pneumonia, is more commonly associated with other diseases, such as septic pleurisy, pericarditis, endocarditis, etc.

Refrigeration, wetting, a preceding trauma, hereditary predisposition, premature mental development, and alcoholism of the parents appear to have a particularly favorable or decisive influence on the development of the affection.

Acute meningitis is of a general or local nature. Predominating upon the convexity, it may also implicate the base of the brain or be present in the latter region exclusively. Here there may be a simple hyperemia of the arterial and

venous vessels of the dura mater and of the pia, or an exudate develops which is serous, sero-fibrinous, or purulent. Quinke has described a purely serous exudate, generally occurring in earliest childhood, which develops with meningitic symptoms and sometimes terminates in recovery after repeated lumbar puncture. Under such circumstances there is more or less decided effusion into the cerebral cavities, lateral ventricles, and even into the third and fourth ventricles. The chambers of the brain are greatly dilated and filled with a transparent serous fluid, which is attributed to a simple serous exudation or an abnormal secretion of the choroid plexus. Quinke believes that vasomotor influences are responsible for the abnormal effusion, which afterward causes an increase of the intracranial pressure upon which the clinical symptoms are dependent.

### SEROUS MENINGITIS

In *serous meningitis* the pia is decidedly reddened and edematous at the base of the brain and upon the convexity, and somewhat cloudy in the neighborhood of the vessels. This cloudiness is not due to leukocytic infiltration, but to a proliferation of the fixed elements. The cortex is usually anemic, the gyri are wide apart, and the sulci have disappeared. This form of meningitis is attributed to cold, mental overexertion, trauma, syphilis, and infectious diseases. Quinke asserts that in most cases there is no infection; other authors mention various microorganisms as causal factors in the pathogenesis of serous meningitis, such as pneumococci, streptococci, bacteria coli, typhoid and influenza bacilli, or their toxins.

In *simple meningitis* the entire surface of the brain, in particular the convexity, is covered with purulent material. The exudate is commonly sero-purulent or purulent and as a rule occupies the arachnoid space. Around the vessels there are slight or extensive purulent infiltrations. In other cases there may also be a sero-purulent or purulent infiltration, more or less extensive, upon the surface and at the base of the brain. The exudate may reach the nerve roots or the optic chiasm at the base of the brain. The ventricles are often free or contain a small amount of sero-fibrinous fluid or similar material, such as is found upon the cerebral cortex. The ependyma is occasionally softened and loose. Capillary extravasations, small suppurative foci, and cerebral abscesses are very rare. The inflammatory areas may be localized in individual areas of the brain, corresponding to the trauma or to disease of the petrous portion of the temporal bone or other carious processes of the skull. The inflammation is often distributed from the membranes of the brain to the meninges of the spinal cord.

The most common microorganisms in the order of their prevalence are the pneumococcus, streptococcus, staphylococcus aureus, the bacillus of Friedländer, the bacterium coli, typhoid and influenza bacilli. The pneumococcus is usually associated with the streptococcus and staphylococcus.

**Symptomatology.**—Upon clinical investigation of the disease in the newborn and in children in the first years of life, we observe general or partial

spasms which recur at short intervals without prodromes, and are accompanied by high fever. Following cessation of the spasm the patient becomes drowsy, somnolent, trembles upon the slightest noise or touch, and shrinks from the light, the muscles of the neck grow rigid, trismus and transitory contractures are occasionally noted, and the child grits its teeth. Strabismus appears, occasionally an ocular nerve is paralyzed, and very often there is transitory paralysis of the face or of the extremities, especially the legs. An important symptom of purulent meningitis, although by no means pathognomonic, to the exclusion of other cerebral diseases, is Kernig's sign: the flexion contracture of the knee-joint which appears when the child is placed upright in bed, or in the lateral posture with the thigh flexed against the trunk. The pulse and respiration are abnormally increased. The great fontanelle is usually prominent. The glance is rigid, the eyes are partially closed, the eyeball is often in constant motion, the pupils are contracted or occasionally show differences in width. The temperature is high,  $40^{\circ}$ – $41^{\circ}$  C. ( $104^{\circ}$ – $105.8^{\circ}$  F.), there is constipation, often vomiting, coma soon appears, and the child perishes with eclamptic attacks or in collapse.

In older children we observe a decided chill, often sudden and without prodromes, with a high elevation of temperature. The cerebral symptoms are quite conspicuous. The patients complain of acute headache, which is increased by looking toward the light, often they cry aloud, and in the first two or three days there is frequent vomiting of bile. Constipation is a common symptom, but is less stubborn than in tuberculous meningitis. Delirium frequently appears upon the first or second day and is associated with audible sighing or increased excitement, or with fear; sometimes it occurs a few hours or a few days after complete repose. During the delirious attack the face is often distorted, the patient cannot swallow, there is retention of urine, rigidity of the muscles of the neck, subsultus tendinum, and when the inflammatory process involves the membranes of the spinal cord there may be abnormal hyperesthesia, rigidity of the vertebral column, occasionally opisthotonos or even trismus. Partial paralyses are rare. The eyeball is sensitive to pressure, the pupils are at first small, but in the later course they show inequality and abnormal dilatation. The respiratory frequency and pulse rate, which at first are increased, become slower and their rhythm is irregular. In the further course the fever is variable, sometimes high, at other times moderate. There may be seeming improvement for some weeks, followed by a sudden relapse and fatal termination. If the patient is lifted from his bed in the first days of the disease and placed upon his feet, the distorted facial expression and the uncertain, waddling gait are conspicuous. When there is no improvement within the first eight days the patient passes into coma and perishes.

**Prognosis.**—Acute meningitis usually terminates in death. Recovery is rare. If the patient does survive there will be defective intelligence or motor paralysis.

*Recovery from meningitis is sometimes observed when the disease occurs in the course of pneumonia or of otitis, and it is therefore obvious that these*



were due to a congestive condition or serous exudation. Secondary meningitis is frequently masked by the symptoms of the preceding disease. The meningitic symptoms may be so slight that they are overlooked. The most severe phenomena of meningitis are presented in that form which develops in the course, or at the onset, of otitis. Occasionally the flow from the ears ceases with the development of the meningeal symptoms, therefore it was formerly believed that if this otorrhea were allowed to take its course the development of meningitis would be prevented. This form of the disease, however, requires quick action and the combat of the pathologic focus while it is localized. Spontaneous recovery is seldom observed. When the outcome is so fortunate it is possible that there was an edema or a serous exudation in the chambers of the brain which originated from the preceding disease of the ear. This is favored by a case of Reicke's, reported by Huguenin, in which, upon the sixteenth day of the disease, copious serous material exuded from the right ear and a profuse diuresis appeared which was followed by cessation of the meningitic symptoms. When an exacerbation ensued, with renewed symptoms of meningitis and a further flow from the ear combined with excessive diuresis, recovery occurred with intact hearing in both ears.

**Differential Diagnosis.**—It must be borne in mind that not until some days after the development of croupous pneumonia, occasionally only immediately preceding the crisis, are we able to determine whether the cerebral symptoms are dependent upon meningitis or are due to the croupous—so-called cerebral—pneumonia. The pulmonary inflammations which appear in the upper lobe, particularly at the apex, or which occur in the axillary fossa, are usually complicated by meningitic symptoms.

Tuberculous meningitis runs a more subacute course. As a rule there are prodromal phenomena which indicate the development of the affection, the fever is less and irregular, delirium is rare, and the course is slower than in the serous type. The anamnesis generally furnishes criteria which determine the nature of the meningitic disease. An accurate history and, most important, a careful examination of the urine, will prevent a confusion of meningitis with uremic convulsions. In children beyond the sixth year hysteria may show a transitory resemblance to meningitis if it is accidentally accompanied by fever, but close observation, the course of the fever, the variation of the symptoms, the presence of other nervous hysterical phenomena, will soon dispel the fear of meningitis.

The diagnostic value of lumbar puncture will be discussed in the description of tuberculous meningitis.

**Treatment.**—The treatment of serous meningitis must be symptomatic and is never to be neglected, even in the most desperate cases. We must consider the sufferings of the patient and do all we can to encourage the parents and relatives and to avert their care and anxiety. We must not lose sight of the fact that meningitis may terminate in recovery. Venesection and strong cutaneous irritants are no longer employed. Lukewarm baths, cold affusions to the head if there is fever, Priessnitz compresses, an ice-bag to the head, calomel in

small doses are the remedial measures to be employed. The preparations of bromin and chloral in enema are indicated for their quieting effect. Inhalations of ether or chloroform should be given for the control of the convulsions. In meningitis resulting from otitis media, cleanliness and thorough disinfection of the auditory canal are necessary, perhaps also trephining of the skull or opening the mastoid process and the antrum. For the otitis a few drops of carbolized glycerin (1-30), or a 3 per cent. boric acid solution should be dropped into the external auditory canal several times daily; when the nose is diseased the cava narium should be cleansed with a 3 per cent. boric acid solution, and borovaseilin applied to the nasal passages. Strict antiseptic measures are to be employed in all external lesions.

### CEREBROSPINAL MENINGITIS

*Cerebrospinal meningitis* is comparatively rare in infancy; in France it is very uncommon, but in other European countries and in America it is of more frequent occurrence. The disease has a sporadic tendency, without being confined to the region of its development. The epidemic appearance, the distribution, and the special symptoms, together with a tendency to recovery, differentiate it in a very conspicuous way from acute meningitis. The malady originates from infection by the diplococcus intracellularis Weichselbaum, which Heubner has designated as the meningococcus intracellularis and which he regards as the only pathogenic agent of the disease. Since 1870 special study has been devoted to the bacteriology of this affection. In 1886 Netter demonstrated that the pneumococcus was frequently the cause of epidemic cerebrospinal meningitis. Weichselbaum, Jäger and Goldschmidt believe the diplococcus intracellularis to be pathogenic of the epidemic form of cerebrospinal meningitis, and Heubner was the first to observe this organism in the living being. In later years Finkelstein, Fürbringer and v. Leyden have demonstrated the organism and Heubner has recently reported that the epidemic form of cerebrospinal meningitis was invariably produced in goats by injections of Weichselbaum's meningococcus in pure culture. This report has been confirmed by many authorities. Heubner states that the meningococcus is less virulent than the pneumococcus, and he thus accounts for the relatively frequent recovery from cerebrospinal meningitis. The affection appears to distribute itself in children usually by contagion, but it also occurs without epidemic influence. The anatomical changes are the same as those observed in the sporadic cases. Upon the surface of the brain we find a thin purulent exudate, and in addition a suppurative infiltration of the membranes of the spinal cord which is most marked in the posterior areas and is localized especially in the cervical and lumbar portions of the cord.

**Symptomatology.**—The symptoms are commonly those of acute meningitis, often associated with acute pain in the course of the vertebral column. The patient complains of pain in the back and in the limbs; the face may be either pale or flushed, vomiting begins early, headache is often so severe that the

patient cries aloud. The pain in the nape of the neck and in the course of the vertebral column is aggravated by movement and by pressure upon the spinous processes. There is also hyperesthesia of the skin. Rigidity of the back of the neck soon appears and may continue until the termination of the disease, occasionally combined with opisthotonos. Trismus, convulsions, sub-sultus tendinum, and even tetanic rigidity are sometimes noted. An eruption of herpes is often noticeable in the region of the face or in other areas and more rarely there are eruptions which resemble measles and petechiæ. The temperature is always decidedly elevated,  $40^{\circ}$ – $41.5^{\circ}$  C. ( $104^{\circ}$ – $106.7^{\circ}$  F.), the pulse throughout is rapid; the patient is extremely restless and soon passes into somnolence and coma. The pupils are contracted, the abdomen retracted, there is incontinence of urine and feces, and the fatal issue frequently appears in a few hours or days. These cases of fulminant course are often the forerunners of epidemics; sometimes there is a remission in the intensity of the symptoms and the disease is protracted for weeks or even months, with slow convalescence. The percentage of recoveries is variously estimated at 25–30 per cent. (Florand), 63 per cent. (Netter), and 68 per cent. Recovery is not always complete. Often there is succeeding paralysis, contracture, deafness, amaurosis, or aphasia, and occasionally there are complications on the part of the serous membranes and the joints.

The **prognosis** in cerebrospinal meningitis is grave, but not so unfavorable as in the other forms.

• **Differential Diagnosis.**—The epidemic distribution, the severity of the individual clinical symptoms, and the microscopic appearance of the cerebrospinal fluid—the presence of meningococci or of pneumococci—are determining factors for the *differential diagnosis* from other forms of meningitis. When the disease is epidemic the patients must be isolated to prevent the possibility of transmission to others.

**Treatment.**—The treatment does not differ from that of other forms of meningitis. The best remedy in the epidemic form is the employment of Quincke's lumbar puncture. A concise description of the therapeutic measures will be given under the treatment of tuberculous meningitis.

The cases which terminate in deafness, blindness, deafmutism, or idiocy must be transferred to suitable institutions.

## TUBERCULOUS MENINGITIS

Tuberculous meningitis is extremely common in infancy. The frequent occurrence, the many obstacles which may arise in the diagnosis, the localization of tuberculosis with its characteristic symptoms, the generally fatal prognosis when the disease has once been recognized as tuberculous, require exact observation and special study on the part of the physician. Moreover, there is the hope that we will eventually succeed in finding ways and means to control this fatal disease which at this time we are almost helpless to combat.

Prior to the middle of the eighteenth century our knowledge of tubercu-



lous meningitis, or acute dropsy of the chambers of the brain, as the disease was formerly designated, was scant.

In 1768 Robert Whytt gave the first accurate description of dropsy of the chambers of the brain in children under two years of age. He divided the disease into three stages, each having individual characteristic symptoms,—a division which is still approved. Whytt arrived at the conclusion that the condition was one of acute internal hydrocephalus. The inflammatory character of the affection was established by Quin (1780) and Ford (1790) in England, Bader (1790) in Germany, and Goelis (1815). The disease was called acute hydrocephalus until 1825, and Senn, who drew attention to its inflammatory nature, upon which the effusion of fluid was dependent, was the originator of the name “acute meningitis.” This author and Guibert had already recognized the granular appearance of the cerebral membranes; Guersant designated the disease meningitis granulosa instead of acute hydrocephalus, but he did not have the temerity to refer it to tuberculous nodules, asserting instead that children who suffered from acute hydrocephalus succumbed because they were phthisical. Guersant’s pupils, Demongeot de Confevron, Tonnelé and Pavoine, Rusz, Fabre and Constans were more exact in their opinion of the tuberculous character of meningitis, but the true condition was only made manifest by Barthez and Rilliet, who differentiated simple meningitis from the tuberculous form and called attention to the distinguishing features of the two diseases. They proved conclusively that simple meningitis develops invariably upon the convexity of the brain, while tuberculous meningitis is (almost always) localized at the base. Meningitis may appear in three forms (Barthez et Rilliet): nodules, and inflammation dependent upon these; tubercles without inflammatory phenomena; solitary tubercle in the brain and meninges. Tuberculous meningitis is often apparently primary, with prodromal symptoms which may be mild or severe, dependent upon a preëxisting latent tuberculosis in other portions of the body, particularly of the bronchial glands; or the affection is first observed in the course of an advanced tuberculosis elsewhere.

Tuberculosis of the serous membranes of the brain is most common in infancy and is rare in adults. It attacks very young children, even infants of five months, and is largely confined to the first five years of life. The period of greatest frequency of tuberculosis of the meninges is variously estimated. Steffen mentions the second year, and Dennig (“Tuberculosis in Infancy,” Leipzig, 1896) states that nurslings offer the greatest contingent. Barthez and Rilliet observed most of their cases in children between three and seven and a half years, while in 159 cases at the Strasburg Children’s Clinic those between the second and fifth years were most frequently attacked. Contrary to the general opinion that boys are affected more than girls, the proportion among my patients treated in the hospital and in private practice is about equal. In my hospital there were 81 girls and 78 boys under treatment. Season appears to exert an influence in so far as the onset of the disease can be determined from the history, the months from October to May furnishing the greatest

number of cases, and of these particularly the spring months. Steffen attributes this to the influence of the damp, cold weather upon the lymph glands and respiratory passages which produces inflammation and catarrh. Predisposing factors are the epidemic diseases, particularly measles, whooping-cough, diphtheria, scarlatina, varicella, and influenza, which are not affected by the seasons. I have not been able to confirm the etiologic importance of onanism and mental stress, which several authors, among them Barthez and Rilliet, D'Espine and Picot, believe to be prominent predisposing causes. *Heredity* is undoubtedly of great influence. In the pathogenesis of meningitis tuberculosa the neuropathic predisposition and alcoholism of the parents play an important rôle. A hereditary neuropathic predisposition is more common among the well-to-do classes than among the poor. Demme, Seeligmüller, Bristowe, Mendelsohn, Hilbert, Schilling, and others, mention a blow or a fall upon the head as causes of meningitis. If importance is to be attached to the statements of Verneuil and his pupils, every injury and every surgical operation would be a predisposing factor of tuberculous meningitis.

Tuberculosis of the meninges is due to the colonization of tubercle bacilli upon the tissue of the pia mater. This is never the result of a primary affection, but there is always disease of neighboring or remote organs. The common starting-point of tuberculosis, is disease of the paratracheal and bronchial lymph glands, or in tuberculous lymph glands of the neck; it may, however, originate from tuberculous affections of the bones and joints, caseous processes in the lungs or in the abdominal cavity, and from diseases of the skin.

By metastasis through the circulation the tubercle bacilli are swept from some old focus over the entire organism, and thus a general miliary tuberculosis develops, tuberculous meningitis being only a part of the prevalent affection.

In other cases the affection is localized in the meninges, or in addition we may find isolated nodules in this or that organ. The development of the affection is then usually due to caseous lymph glands, particularly the bronchial glands.

However, a primary caseous tuberculous focus is not always apparent post mortem. We must then assume that it was not found at the autopsy and that some bronchial gland of normal external appearance contains Koch's tubercle bacilli, from which the affection started (Loomis and Pizzini).<sup>1</sup>

The results of these investigations lack confirmation.

The infectious focus may also lie in the vicinity of the cerebral membranes; the disease may originate in the skull from caries of the cranial vault or of the petrous portion of the temporal bone, or be due to solitary tubercle in the brain; further, it may arise in the nasal cavity, in the auditory canal or

<sup>1</sup> Martens, "Traité des Maladies de l'enfance publié sous la Direction de Mm. Grancher, J. Comby et A. B. Marfan."—Loomis, "Researches of the Loomis Laboratory," 1890, cited.—Pizzini, "Tuberkelbacillen in den Lymphdrüsen Nichttuberculöser." *Zeitschrift für klinische Medizin*, 1892, Bd. 329.

from adenoid vegetations which contain tubercle bacilli. The infection of the meninges may occur by direct transmission through the lymph tracts which surround the nerves at the base of the brain.

**Pathologic Anatomy.**—Tuberculous meningitis may appear in various forms. Most frequently the tubercles are distributed in the course of the vessels; the cortical and medullary substances of the brain are rarely affected. When the brain is removed from the skull we note that the surface, the pia mater, has a more or less hyperemic appearance. At the base of the brain in particular there are meningitic changes. Tuberculosis is usually bilateral, but it is often more markedly developed upon one side, especially in the region of the arteria fossæ Sylvii.

In addition to nodule formation there are serous, sero-fibrinous, and gelatino-purulent exudations in the subarachnoid space, in the brain substance itself, and in the sheaths of the pia following the ventricles. With these changes the disease may be designated, following Robert Whytt, *meningo-encephalitis tuberculosa*. If the inflammatory exudations are not present, or are slight, which is uncommon and occurs mostly in cases with a chronic course, it is better to speak of tuberculosis of the meninges. The gelatino-purulent exudate is particularly abundant around the vessels, actually enveloping them; it surrounds the cerebral nerves, and completely occludes the spaces between the cerebral convolutions, especially the incisura fossæ Sylvii. The superior vermis of the cerebellum and the upper portion of the medulla oblongata are often surrounded by a greenish, purulent exudate; the exudation is usually most copious in the region between the pons and optic chiasm.

Tubercles in the pus which covers the pia mater are frequently difficult to distinguish and are often not perceptible until the membrane has been sprayed with a slow stream of water. The tuberculous nodules are most abundant in the course of the vessels and in the lymph tracts, particularly at their points of bifurcation. Sometimes distinct gray or yellowish nodules, exceptionally caseous masses, are found deep in the sulci. Microscopic examination reveals great numbers of tubercle bacilli in these nodules, also in the perivascular lymph tracts and in the central coagula of blood. They are also found in the exudate, and in one case of tuberculous meningitis Heubner found meningo-cocci twice and pneumococci once in the fluid obtained by lumbar puncture.

Under the infiltrated pia the brain substance shows focal or widely distributed softening, is reddened, and often stained by a small effusion of blood. Frequently there is molecular destruction of the cellular elements of the neuroglia, which leads to destruction of nerve fibers and of the ganglion cells.

If acute hydrocephalus develops the substance of the brain is softened and edematous, and the veins show injection. The amount of ventricular fluid varies proportionately between 20 and 100 c.c. The fluid is most copious in the lateral ventricles, but is also found in the third and fourth ventricles, sometimes clear, occasionally turbid, with admixture of fibrin flocculi; it is seldom purulent. Under treatment, especially in the discussion of lumbar puncture, I shall recur to the composition of the ventricular fluid.



The choroid plexus is reddened, swollen, enlarged, and sometimes softened. The changes of the choroid plexus eventually play a rôle in the development of hydrocephalus. By pressure of the ventricular fluid upon the substance of the brain the gyri are separated, the sulci obliterated, the subarachnoid fluid is expressed, the cortex of the brain becomes anemic, and the arachnoid surface dry.

Isolated nodules are also found in the convexity, upon the pia. Occasionally numerous nodules with a gelatino-purulent exudate are observed in the pia sheaths, now and then with small, localized foci of inflammation or softening in the brain. The convexity of the cerebellum may also be involved. The dura mater is often permeated with tubercles, particularly at the base of the skull over the sella turcica. In the convexity the tubercles are always on the inner surface of the dura mater. Exceptionally, under the influence of the tuberculous infection there is a development on the dura mater of pseudo-membranes which are permeated by hemorrhages. Larger tubercles are not uncommon. In some cases they are altogether absent and there is merely an inflammation of the pia. In tuberculous meningitis the membranes of the spinal cord are usually involved. According to v. Leyden the greater development of the inflammatory process on the posterior surface of the spinal cord membranes is due to gravity, but Erb "regards this preference of the posterior surface of the cord as due to the numerous meshes and septa of the posterior subarachnoid space which have been observed by Axel Key and Retzius." The essential reason therefor is the direct relationship of the arachnoid sac of the brain and spinal cord (v. Leyden), and the constant movement of the cerebro-spinal fluid, as was demonstrated experimentally by Quincke, is the factor which propagates the inflammation from the brain to the spinal cord and *vice versa*.

The spinal cord is often involved in the same manner as the brain, in that the tuberculous inflammatory process, through the agency of the pia, which sends its branches to the nerve substance beneath, is propagated to the spinal cord. But it also appears that the tuberculous affection of the meninges is dependent upon tuberculosis of the vertebra and is then distributed from the membranes of the spinal cord to the meninges of the brain. The emerging nerves and the nerve roots are often involved. Microscopic examination reveals that we are dealing with the development of nodules or with a tuberculous infiltration of the pia spinalis, with frequent implication of the spinal ganglia. The axis cylinders are sometimes swollen, sometimes they show granular degeneration, the medullary sheaths are degenerated, the spinal cord shows in some cases a beginning acute interstitial myelitis (Schultze). This explains certain of the symptoms of meningitis, such as hyperesthesia, anesthesia, and paralysis. Changes in the eye-ground are common, as well as venous hyperemia of the retina and optic neuritis with terminating atrophy. Choroid tubercles are rarely observed.

**Symptoms and Course.**—Tuberculous meningitis is either a partial phenomenon of general miliary tuberculosis or the meningitic symptoms develop with cerebral phenomena and without tuberculous involvement of other or-

gans. This form of the disease occurs principally in childhood and is to be the subject of our further discussion.

The symptom-complex of tuberculous meningitis has been divided by various authors into definite periods. Thus R. Whytt proposes three divisions, regulated by the condition of the pulse. Goelis distinguishes a stage of turgescence to the head, of local inflammation of the membranes or brain substance or of both simultaneously, of transudation following inflammation, and of paralysis. Plenck and Henke designate the first period "febrile," the second "apyretic," the third "lethal." Among more recent authors Steffen, following Traube, divides the course of the disease into a stage of irritation, of pressure, and of relaxation. Vogel and Henoch assert that an attempt at such a division is futile, as there are too many deviations from the typical course. It is well to omit a division into definite periods and, like Henoch, to merely differentiate the stages of irritation and of paralysis.

The character of the pulse follows a definite rule throughout, therefore, following the arrangement of Whytt (Rilliet et Barthez, Votteler), I shall next give a description of the individual symptoms as they arise prior, during, and subsequent to the period of slow pulse.

The first of these is *headache*, which in the prodromal stage is not persistent. The head is grasped with the hands or inclined to one side, the facial expression denotes pain, and the child often moans or cries, "My head! My head!" Older children complain of headache now and then, which may be so intense that they cry aloud; the pain may appear in paroxysms and be exacerbated by intense light. As a rule it is localized to the frontal region and either disappears with increasing stupor or the patient is less conscious of it. Frequent crying and grasping of the head apparently denote the presence of headache even in the somnolent condition. The pain ceases, but constantly recurs in varying intensity. Headache may be regarded as the expression of an increased intracranial pressure (Emminghaus), the arachnoid portions of the pia (after displacement of the minimal quantity of subdural fluid, which was demonstrated by Hitzig) being pressed toward the fibrinous layer, which Hitzig claims is rendered highly sensitive by the ramifications of the trigeminus and pneumogastric nerves. As a rule, at the beginning of the disease infants do not take the breast so regularly as in health, the patient emaciates and the skin becomes pallid and wrinkled, notwithstanding the sufficient ingestion of food; in other cases nourishment is taken irregularly. The child soon lets go when put to the breast, falls asleep, cries upon renewed attempts to make it suckle, and soon slumbers again.

The tongue is sometimes coated, and diarrhea alternates with constipation. Sleep is restless, often interrupted by sudden outcries. The eyes are languid, dim, the countenance denotes suffering, occasionally the child cries aloud; but not until the appearance of more serious symptoms, such as severe headache or convulsions, is the physician consulted. The whole nature of older children is frequently changed. Those who are naturally active become quiet, ill-humored, and protect the head or bury it in the mother's lap. Others are fidgety

or sit in a corner, feel too weak to walk, mope, become listless, and refuse to take part in the games of their playmates. They are depressed, fretful, dissatisfied with everything, cry upon the slightest provocation, are disagreeable, quarrelsome, and disobedient. Sleep is usually restless and oftentimes interrupted by fright or loud outcries. At school these children are listless, confused, and cannot follow the instruction. Appetite is usually poor or irregular, the bowels are often constipated, or there may be diarrhea. These conditions are frequently well developed; again they are insignificant and raise no suspicion of the gravity of the affection until a difference in the pupils, a transitory strabismus, and increasing pain cause the parents to consult a physician. The *prodromal symptoms*, which are of varying intensity, are referred to hyperemia of the pia brought about by miliary tuberculosis. The affection may, however, develop in quite the same manner as epidemic cerebrospinal meningitis, with high fever and slow pulse, or the pulse may be abnormally rapid or intermittent. The child passes rapidly into stupor and coma with continued high fever, and life is soon extinguished. Cases are on record which terminated in ten hours (Dennig), in two hours (Rohrer), and in four and one-half hours (Seeligmüller).

The onset may be acute and followed by a cessation of the symptoms for several weeks (Dennig); then the cerebral symptoms appear and the case terminates in death.

*Vomiting* usually accompanies the headache and general symptoms. It occurs independently of the ingestion of food and is sometimes observed as often as seven times in the course of the day. It appears abruptly, without preceding nausea, and is copious, or it may be superseded by retching. Vomiting is induced by quick elevation to the sitting posture and also after ingestion of fluid. The vomitus is often bile green, sometimes admixed with mucus and at first with particles of food. This symptom appears with the early meningitic phenomena, continues one or more days, then usually disappears and is rare after the first week. Vomiting is by no means pathognomonic of tuberculous meningitis, but when it appears suddenly and without cause it occupies an important position in the diagnosis in connection with the history and the accompanying symptoms. It is then probably due to an irritation of the vomit center in the medulla oblongata (Hermann, "Physiology," p. 136) which is produced by hyperemia, reflex anemia, or other influences.

*Constipation* occurs simultaneously with vomiting, or precedes it, and fails to yield to the strongest purgatives. This sluggishness of the intestines is almost exclusively peculiar to meningitis, and is observed together with *scaphoïd* (retracted) *belly*,—one of the most important phenomena of tuberculous meningitis. The cause of the constipation has not been determined. It is probably due to an irritation of the splanchnic nerves (Hench, Barthez and Rilliet)—the nerves of inhibition of intestinal movements; the peristaltic movements are very sluggish or are difficult to detect under the emaciated cutaneous coverings of the belly. The vomiting, constipation, the retraction of the abdomen probably have a common pathogenesis. Vogel is of the opinion



that the retracted belly is the result of a constant pathologic contraction of the transverse and oblique muscles. Very few of my cases showed a contraction of these muscles.

Slight, irregular *fever* is present from the beginning of the disease, the temperature rising in some cases to 39° C. (102.2° F.) in the late afternoon hours. Now and then, however, the elevation is higher, 40° to 41° C. (104°–105.8° F.), and remains so throughout the twenty-four hours. The pulse is at first rapid and irregular, with quick fluctuations both in frequency and fullness. After the introduction of cerebral symptoms, as somnolence, the pulse is slower—60 to 80 beats—and of irregular rhythm, sometimes intermittent. The slight movements of these patients, the turning in bed, and also mild psychical irritation, are sufficient to stimulate the pulse frequency materially, the pulsations increasing to 140–160 per minute.

To these symptoms *photophobia* must be added. The patients are apathetic, close their eyes, turn their faces to the wall, and are annoyed by all sounds. When questioned by the physician or relatives they grow restless, turn away in fear, or moan and cry aloud. They whimper and frequently exclaim, "My head! My head!" and grasp some portion of the head with their hands. There is hyperesthesia of the skin and the muscles; mild cutaneous irritation is sufficient to provoke crying. Pressure upon the eyeball is painful. Although sleep is essential, it is an impossibility. In a half-awake condition they rise suddenly, sit upright in bed and gaze about. Delirium is rarely an accompaniment of this symptom in children. From time to time deep sighs are heard. The apathetic condition passes gradually into somnolence, interrupted frequently by loud crying (*cris hydrencéphaliques*, Coindet), the eyes are partially closed with the bulbi rolled upward and inward. When called loudly the patients show but slight consciousness or give brief or unintelligible replies and lapse again into stupor. They then assume the so-called "*hunting-dog posture*": the trunk is inclined forward, the thighs are drawn up against the lower abdomen, the back of the neck is stiff, occasionally there is a well-developed *rigidity of the neck*.

The appetite fails, fluid nourishment only is taken, there is no apparent increase of thirst, the tongue is coated. Emaciation is rapid and extensive. When somnolence is well marked the patients refuse food, and if trismus develops, with an arrested deglutition reflex, the only recourse is artificial nourishment with a stomach tube. The urine is scant, but free from albumin. The liver and spleen are occasionally enlarged.

During the *second week* of the disease the entire pathologic picture is changed, together with the disappearance of vomiting and of severe headache. The fever decreases, sometimes to the normal temperature. The pulse becomes slower and is irregular. During examination of the pulse we must palpate cautiously, for any excitement is sufficient to send the pulse from 60 to 140, or even 160, beats per minute. *Such abnormal slowness of the pulse in a febrile disease is noted only in tuberculous meningitis [and yellow fever]*. Respiration is irregular; deep, long-drawn inspirations are followed by those of

short, superficial, rapid character. The breathing may cease and begin again with a deep sigh. Deep and shallow breathing alternate. Occasionally the Cheyne-Stokes type prevails—the respiration becomes slower, intermittent, and interrupted with sighs.

*Vasomotor* symptoms may be noted. The color of the face shows great variation. Flushing and pallor alternate rapidly. Large red splotches may suddenly appear on the face, increase in size, and in a short time vanish. Slight cutaneous irritation with the finger or a pencil drawn over the surface at once produces red welts in the path of irritation, which distribute themselves and after a while disappear. Trousseau, who first described this phenomenon, erroneously regarded it as pathognomonic of meningitis and designated the symptom "*tâche cérébrale*" or "*méningitique*." Exanthems are rarely observed in tuberculous meningitis, although urticaria, roseola, and other eruptions resembling measles are sometimes seen. Herpes labialis and herpes zoster are occasional phenomena. In the course of the disease purulent eczemas not infrequently dry, profuse secretion of the nasal mucous membrane ceases, and the early diarrhea disappears (Henoch). The swelling of the cervical glands is reduced, often within a few days, under the influence of meningitis (Henoch). Occasionally there are profuse outbreaks of sweat, perhaps unilateral, and for a few days prior to death the face may be bathed in perspiration.

*Ocular changes* are conspicuous from the outset. Even in the first days of the disease the luster of the eye peculiar to children is lost, the glance is often fixed, dreamy, and when somnolence appears it is expressionless. Photophobia is common. The eyeball is sometimes rolled upward and inward, and there is convergent strabismus, which vanishes when the child is recalled to consciousness by loud talking—a condition, however, which Rählmann and Wittkowski claim is not unusual in childhood during sleep. Divergent strabismus may also be present. Whether or not there is double vision cannot be determined because of the involvement of the sensorium. The strabismus is usually transient. Incomplete closure of the lids and anesthesia of the cornea due to paralysis of fibers of the fifth nerve cause an over-exposure of the sclera and cornea to the air and a consequent dry appearance, and the eye may be covered with a thick conjunctival secretion. Later the cornea becomes uneven, clouded, but is rarely ulcerated or destroyed. Toward the end of the affection the sclera shows injection of the venous vessels. At first the pupils are usually contracted, later they are dilated and often reveal a transitory inequality. Without apparent cause a normal pupil will be widely dilated and subsequently show decided contraction. Pupillary reaction, although slow, is generally retained. When cutaneous irritation is marked a transitory dilatation of the pupils is frequently produced by pinching the skin. Now and then there is nystagmus, the movement of the eyeball being undulated and oscillatory, in other cases these movements are spasmodic, rhythmic, occurring about 70 times a second. With the development of rapid pulse the pupils commonly assume their normal width; they may, however, be markedly con-

tracted, especially in deep coma. The reaction to light is then usually lost and a ray of light near the eyes fails to induce closure of the lids or pupillary reflex. Neither the retina nor the optic nerve can be stimulated. Amblyopia and amaurosis appear.

During general convulsions the pupils are dilated and in the intervals they contract. The origin of pupillary changes is very complex and it is difficult to determine whether the sympathetic or the oculomotor nerve exerts alternately a stimulating and paralyzing influence upon the width of the pupils. The irritative and paralytic phenomena may be due to an increase of the intracranial pressure and to direct inflammation, and in that case the nerve centers or the nerve fibers are affected in their course.

Ophthalmoscopic examination seldom reveals choroid tubercles.

The eye-ground is often normal. In other cases we find slight hyperemia of the eye-ground with a normal, clearly defined pupil, or venous hyperemia of the retina and often slight choked disc. Optic neuritis, occasionally terminating in atrophy, may also be observed.

The explanation of the inflammation of the papillæ may be found in the relation of the arachnoid to the subvaginal space (Schwalbe), and Virchow was able to demonstrate perineuritis and interstitial neuritis in the course of the optic nerve. As to the diagnostic value of the ophthalmoscopic findings I may state that changes in the eye-ground are usually not conspicuous until other symptoms have arisen which unquestionably indicate tuberculous meningitis.

In doubtful cases running an abnormal course ophthalmoscopic investigation may occasionally be of assistance in the diagnosis.

During the second week *irritative phenomena*, transitory *convulsions*, and general or partial *contractions* appear. Muscular contractions of the face and of the eyelids, nystagmus, trismus, movements of sucking and of mastication, gritting of the teeth, or tonic or clonic spasms of the extremities are observed. In the last condition special muscle groups are implicated or the entire body is involved. The spasms are of varying intensity; they may continue for an indefinite time, then cease for a few hours or days, and begin anew. During the intervals there may be contracture in various muscle regions, particularly of the adductors of the thigh and the flexor muscles of the upper arm. Picking of the bedclothes is also common. In addition there is marked rigidity of the neck, the head is frequently burrowed deeply into the pillows, opisthotonos may be present and is sometimes so extreme that the stiffened body can be raised by pressure of the hand under the head.

*Rigidity of the neck* is always associated with pain over the cervical vertebræ induced by pressure. Occasionally there is a relaxation of the muscles of the neck which permits movement of the head, but the painfulness continues. The younger the child the earlier is this symptom apparent. In regard to the nature of this rigidity I must remark that according to v. Leyden it is the consequence of an implication of the medulla oblongata or of the upper portion of the cervical cord, not, however, of spinal meningitis. Inversely



opisthotonos is a symptom peculiar to diseases of the spinal cord membranes dependent upon contracture of the muscles of the back. Opisthotonos is particularly prominent in cases which run their course with high fever. Rigidity of the neck and of the vertebral column frequently vary in intensity, sometimes to a complete relaxation of the contracture. External irritation may reproduce the opisthotonos. This symptom is always associated with contractures in the lower extremities, particularly with rigidity of the adductors of the thigh, and sometimes with contractures of the muscles of the arms. Coma is present in all cases. Occasionally the hydrocephalic cry of Coindet is heard. This is not due to a conscious sensation of pain, but is purely automatic (Trousseau).

In this period of the disease the patient lies quiet, the fever abates or disappears, the pulse rate is decreased, the headache and hyperesthesia are less intense, and vomiting has ceased. Those around the patient believe that he is better and hope is awakened that his life will be spared. The experienced physician, however, will not be misled by this apparent improvement.

In the *terminal stage* sopor or coma is combined with paralysis or convulsions. The last condition occasionally is limited to an extremity, often a mere contraction, while in other cases general spasms are observed. In addition frequently there are paralytic symptoms, usually mild, which involve one or another extremity and are sometimes associated with paralysis or paresis of the facial, the oculomotor, or the abducens nerves. Paralysis of the facial or of the ocular nerves, however, is frequently observed without simultaneous paralysis of the extremities. This symptom is largely due to an exudate surrounding the nerves at the base of the brain. Occasionally the necropsy shows no distinct changes. In tuberculous meningitis, however, there are *permanent* paralyzes due to *focal disease* of the brain, softening, obliteration of the arteries, or to solitary cerebral tubercle. According to the localization we find hemiplegia, monoplegia, or crossed paralysis.

The patients lie in bed with eyes widely opened, or the glance is half veiled. The pupils, which were previously dilated, contract and do not react to light. There is amaurosis, the patient no longer shows consciousness, reacting neither to a call nor to mechanical stimulation. The sudden changes in the color of the skin are conspicuous; flushing and pallor rapidly alternate. Although these symptoms are present in the first weeks of the disease they do not become prominent until toward the end of the second or early in the third week, or at the close of the second or the beginning of the third stage. The skin is shrunken and dry, the adipose tissue indicates wasting, and at times an erythema appears over the entire body.

Contraction of the abdomen becomes so prominent that the abdominal covers almost lie upon the vertebral column. Toward the end the temperature of the skin increases; in the rectum it may register 40° to 43° C. (104°–109.4° F.). The pulse is rapid (160–200) and with decrease of cardiac power marked cyanosis appears, the skin is often covered with cold, clammy perspiration, the respiration is hurried and irregular, sometimes of the Cheyne-Stokes type,

feces and urine are voided involuntarily, and gradually, perhaps with preceding general spasms, life ebbs away. The post mortem temperature is almost always high, and in the rectum may be 43° C. (109.4° F.).

This is the general picture of tuberculous meningitis. The disease, however, presents no uniform course, one or another characteristic symptom often being absent. The headache and vomiting may be slight or fail to appear; fever may not occur until toward the end of the disease; the convulsions, paresis of the oculomotor, abducens and sympathetic nerves need not occur; but the nature of the disease is evident to the skilled physician without a complete symptom-complex.

A further condition which is often found at the onset of the affection in nurslings and in children during the first two years of life is eclampsia, associated with somnolence, which may again be interrupted by renewed attacks of spasm, sometimes accompanied from the onset by temporary rigidity of the neck. This condition is succeeded by a rapidly developing coma and death follows in the course of three or four days. In other cases, especially in adults and in older children, the meningitic symptoms arise suddenly in the course of well-advanced pulmonary tuberculosis or tuberculosis of other parts of the body, especially of the bones. In adults this condition is frequently associated with delirium—a rare phenomenon in children—and the patient perishes in a day or two.

*Partial meningitis* may develop primarily or as a secondary affection. The symptoms are similar to those of general meningitis, combined with other phenomena such as spasms and paralysis which are dependent upon the seat of the disease. Here we observe convulsions, various paralyses of the eyes and face, monoplegia or paresis, hemiplegia and paraplegia, with and without contractures. The development of these paralytic phenomena may be slow or apoplectiform and is the result of analogous conditions such as are noted in solitary tubercle of the brain.

The *membranes of the spinal cord* are involved in most cases of tuberculous meningitis. The spinal symptoms, however, are dominated by those of basilar meningitis. The patients occasionally complain of pain in the lumbar region and of a disagreeable, tormenting sense of constriction as though the thorax were compressed and the chest bound with hoops [girdle pain]. Radiating pains are sometimes felt in the lower extremities, with hyperesthesia of the abdominal walls, muscular contractions, and subsultus tendinum. Marked rigidity of the vertebral column, opisthotonos, increased reflexes, and even a complete tetanus are observed. In the later course, after the pains have lessened, the paralytic phenomena of the lower extremities and of the bladder predominate.

**Prognosis.**—Tuberculous disease of the meninges offers a very serious prognosis. Quite exceptionally there may be an arrest of the tuberculous process, or there may be a prolonged quiescence of the symptoms, but after a few weeks, infrequently after months, the symptoms of meningitis recur and the patient succumbs. When there is actual recovery the doubt always remains whether we were dealing with true tuberculosis.

In some patients who present the symptoms of tuberculous meningitis the acute inflammatory phenomena occasionally subside and there may be, as we know, calcification and encapsulation of the tuberculous focus, and infiltration, but the further consequence of these chronic changes manifests itself in the form of idiocy and epileptic attacks (Marfan). A few authors have noted recovery of cases in which tubercle bacilli were demonstrated microscopically in the lumbar fluid (Leube, Freihan, M. Henkel). As to the value of lumbar puncture in tuberculous meningitis, we must admit that an arrest of the tuberculous process in the membranes of the brain may occur in isolated cases of tuberculous meningitis even without therapeutic aid, as is now and then demonstrated at the post mortem table after the lapse of years.

In my hospital and private practice not one of the cases in which I have made a diagnosis of tuberculous meningitis has been saved even by lumbar puncture.

**Diagnosis.**—The diagnosis does not cause much perplexity. A careful history, a minute examination and cautious observation of the course of the symptoms, will protect us from error. As has been already explained, even though one or another symptom may be absent, the individual phenomena eventually group themselves into a pathologic picture which permits a positive diagnosis of tuberculous meningitis. The gradual development of the disease, early irregular fever which soon terminates with a fall of temperature to normal and below, the condition of the pulse, its retardation and irregularity and the succeeding abnormal rapidity which sets in at the end of the second or the beginning of the third week, the severe headache, the paralysis in the course of the ocular and facial nerves, the inequality of the pupils, the somnolence which appears later, the spasms, the coma are sufficient to determine the diagnosis.

Certain difficulties may arise in the differentiation from febrile gastric affections which at their onset occasionally simulate meningitis. Children complain of headache and pain in the abdomen, there is vomiting and constipation, the appetite is poor, the tongue coated, and often there is conspicuous photophobia. In addition there may be irregular fever, ranging from 38° to 38.5° C. (100.4° to 101.3° F.), and increased but regular pulse. Such affections usually begin acutely and may often be attributed to indigestion. The patient is fretful and cries, and the physician may suspect the development of severe meningitis. The further course, however, will reveal the correct diagnosis. In a simple febrile gastric catarrh the pulse is not retarded and irregular, as in meningitis, but remains rapid and small, although in convalescence, when all other symptoms have disappeared, a certain transitory irregularity may be noted.

*Enteric fever* sometimes presents the symptoms of meningitis, now and then combined with aphasia and unilateral paralysis. If it is doubtful whether we are dealing with enteric fever or meningitis an examination of the blood for the Widal reaction, and lumbar puncture, will decide the question.

*Other forms of meningitis* which appear in the course of infectious diseases, or owe their origin to otitis or to affections of the nose or pharynx, can



readily be differentiated if we take into consideration the foregoing disease and carefully examine the ears and the nasal and pharyngeal cavities, which will reveal at once whether we are dealing with a tuberculous or other form of meningitis.

Cerebrospinal meningitis, in contrast with meningitis tuberculosa, is usually of acute onset, without prodromes, and with high fever. The spinal symptoms are most prominent—abnormal rigidity of the vertebral column, contractures of the muscles of the extremities, tremor, subsultus tendinum—and the disease usually presents an epidemic tendency from the beginning. Lumbar puncture may enlighten us as to the nature of the meningitis. According to the investigations of E. Bendix (*Deutsche klin. Wochenschr.*, 1901, No. 43) the cytologic quality of the cerebrospinal fluid furnishes a positive means of differentiation of tuberculous from purulent or epidemic meningitis. In five cases of tuberculosis of the meninges which were treated in Prof. Minkowski's wards in the Augusta Hospital in Cologne it was invariably noted that the cellular elements of the cerebrospinal fluid consisted largely of small, mononuclear pus corpuscles—lymphocytes—and that the large polynuclear leukocytes were scarce. Inversely, in purulent or epidemic meningitis the leukocytes preponderate.

The differentiation of meningitis simplex from meningitis tuberculosa may occasionally be perplexing. The acute development, the convulsions which often appear at the onset of the disease, and the uniformly high fever favor the former affection. Tuberculosis of the meninges may exceptionally present the same clinical picture as simple meningitis, therefore when the patient's relatives report trauma as the cause of the disease, and a hereditary predisposition cannot be established, the differential diagnosis for the time being is impossible. The presence of tubercle bacilli in the lumbar fluid is a positive diagnostic factor.

The differential diagnosis of tuberculous from syphilitic meningitis in children as a rule cannot be made. The favorable progress of the disease following the employment of mercurial preparations, calomel, and inunctions would indicate the syphilitic form.

Symptoms suggestive of meningitis may occasionally be noted in helminthiasis. An accurate history and careful observation will protect us from error, and if there is a suspicion that such symptoms as vomiting, headache, vertigo, fretfulness, irregular pulse, etc., are brought about by the irritation of worms, the administration of santonin or other suitable vermifuge, together with calomel, will soon give us an insight of the nature of the affection.

**Treatment.**—Tuberculous meningitis must be designated as an incurable disease. I have never seen recovery, and the cures reported by other authors (among them Politzer, Bókai, Henoch) were soon followed by relapse.

It would, however, be inhumane to at once predict this unfavorable outcome to those about the patient, as errors may be made in diagnosis; we must not discourage all hope of a favorable termination, and a positively lethal prognosis should be withheld until we find that the patient is passing constantly from bad to worse and that the only relief will be death. We must not content

ourselves with therapeutic nihilism. Calomel may be given in small doses for relief of the constipation; inunctions with unguent. hydrargyri should be tried. If, however, there is an eventual favorable result we will have to admit that we were not dealing with tuberculous meningitis, but with a meningitis of syphilitic origin. According to Quinke mercurialization by inunctions with blue ointment, which should be continued from four to six weeks, is very beneficial in the serous form of the disease.

The common therapeutic measures, as leeches, ice, and calomel, have no noteworthy influence, and this is also true of resorbents and derivatives. Inunctions to the nape of the neck or upon the scalp with strongly irritating ointments such as tartar emetic ointment, unguent. veratrinæ, guaiacol ointment 10–15 per cent., and painting with tincture of iodine are more annoying to the patient than efficacious.

The treatment must be purely symptomatic and directed principally to the alleviation of pain, decrease of restlessness and of the irritative phenomena, the relief of spasm with its accompanying pain, and the control of constipation; further, we may attempt to reduce the pyrexia and administer sufficient nourishment, so far as this is practicable.

The application of an ice-bag to the head occasionally lessens the pain; but if this cannot be controlled, small doses of morphin or codein may be given, or hypodermic injections of morphin up to 4 Mgs. For the relief of severe spasms enemata of chloral hydrate are of service (0.05–0.5–1.0 to 50 of distilled water or decoction of althea). If there is high elevation of temperature baths at 34° C. (93.2° F.) and gradually reduced to 30° C. (86° F.) by the addition of cold water, are indicated. The mouth should be carefully cleansed and if conjunctivitis and keratitis develop, a protective dressing is absolutely necessary. Nutritive enemata are sometimes useful.

For retention of urine warm fomentations may be applied to the abdomen, or the bladder should be emptied by catheterization. In conditions of collapse such as fall of temperature, marked cardiac asthenia, and coma, warm baths with cold affusions are indicated. Thus far no favorable result has been observed from injections of tuberculin.

Nothing is to be expected from trephining of the skull or puncture of the lateral ventricles.

On the other hand we should endeavor to bring about amelioration, or recovery perhaps, by *lumbar puncture*, which was introduced by Quinke in 1891 and is carried out according to his well known directions.<sup>1</sup> This is generally performed in the third interarcual space between the third and fourth lumbar vertebræ, in either the right or the left lateral position, with the body inclined forward, the knees flexed against the abdomen so that the vertebral column is curved and the spinous processes are prominent. The needle is inserted in the median line to a depth of 2 to 4 cm., according to the age of the child. If fluid does not appear the needle may be directed

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<sup>1</sup> See article on Lumbar Puncture by Quinke in the volume on "Diseases of the Nervous System," p. 223.

slightly upward. I have never seen sudden death follow these maneuvers, nor have I observed serious consequences such as hemorrhage, convulsions, deviation of the needle, false passages. The quantity of fluid obtained by puncture varies from 4 to 100 c.c. (*Therap. Monatsh.* 1900.) The pressure ranges from 180 to 750 Mm. water.

The value of lumbar puncture depends primarily upon whether the pressure is increased and the quantity of fluid is excessive. The amount of albumin contained in the cerebrospinal fluid in these cases shows an average variation of  $\frac{1}{4}$ ,  $\frac{1}{2}$  and 1 per 1000; in one instance it was  $2\frac{1}{2}$  per 1000. I have never found sugar. The specific gravity ranges between 1002 and 1008. The fluid is usually clear; occasionally I have seen a slight turbidity. After standing a few hours the water-clear fluid sometimes shows a slight floccular precipitate. The tubercle bacillus is not always demonstrated upon bacteriologic investigation. According to the reports of various investigators this microorganism is found in 50 to 80 per cent. of the cases. In general miliary tuberculosis, in solitary cerebral tubercle, and in general tuberculosis of other organs, the cerebrospinal fluid will very often be found sterile. I do not attach much value to the presence of tubercle bacilli in the lumbar fluid, as in tuberculous meningitis the entire course, the character of the fever, and the basilar brain symptoms are sufficient for a positive diagnosis without the employment of lumbar puncture or an examination of the eye-grounds (choroid tubercles). Bacteriologic investigation of the lumbar fluid is of great importance in determining the nature of the meningitis, whether tuberculous or suppurative.

Some therapeutic value may be attached to lumbar puncture from the fact that for a brief period following this slight operation the patient occasionally awakens from coma, sits up in bed, asks for food and drink and shows a transitory interest in his surroundings. Nevertheless there is an eventual relapse. Spasms may cease and we gain the impression of a decided improvement. I have never been able to demonstrate a permanent benefit from lumbar puncture in tuberculous meningitis, and although some authors (J. Schwalbe, Leube, Freihaan, Henkel) have reported recovery following that procedure, nevertheless when we consider its therapeutic influence we must remember that in very exceptional instances an arrest of the tuberculous process in the cerebral membranes may take place even without therapeutic aid, as is demonstrated now and then at the post mortem table days and weeks or even years after the condition had occurred. However, taking into consideration the transitory relief by lumbar puncture of the most distressing symptoms (delirium, pain, convulsions, outcries), I would always favor this method. It relieves not only the patient, but those about him; the relatives again hope for improvement and feel that help is still available; it saves the physician from complete powerlessness in this otherwise dreadful and fatal disease, and to some degree at least alleviates the suffering.

I have seen favorable results from lumbar puncture in purulent cerebrospinal meningitis. The cerebrospinal fluid contained meningococci or diplococci. Of six patients upon whom I performed lumbar puncture for suppura-



tive meningitis two were cured, the third case, apart from complete deafness, was discharged from the hospital quite well, the fourth, a weak, rachitic child, recovered from meningitis, and showed normal bodily development but was deficient mentally and is not yet able, although five years old, to stand alone or walk. These favorable results coincide with those of Netter, who reported recovery in 7 out of 11 cases of purulent meningitis, the diagnosis having been made by lumbar puncture.

Recovery occurred as a rule in five to fourteen days, but in some cases required two or three months.

Netter repeated lumbar puncture in his patients from one to ten times. He believes that the favorable result of puncture is reinforced by the employment of hot baths of twenty to thirty minutes' duration, at a temperature of 38° to 40° C. (100.4° to 104° F.), repeated every three or four hours day and night. I have observed no noteworthy influence upon choked disc, as has been reported by some authors, among them Landolt (Strasburg). Attention must be devoted to the nutrition of such patients, and if there is decided loss of strength subcutaneous infusions of normal salt solution are indicated.

In serous meningitis the views as to the benefit of lumbar puncture are widely diverse. Quinke, Heise, Henschen, and v. Leyden report good results; on the other hand Fürbringer, Pott, and Fleischmann have seen no favorable effect from lumbar puncture and have known disagreeable symptoms to follow the procedure. Nevertheless this slight operation should always be performed, and I must particularly advise its repetition after a few days, with proper anti-septic precautions, and that each time only a small quantity of fluid, 30 to 50 c.c., should be withdrawn.

In chronic cases a slitting of the dura mater by means of a long-handled knife with a blade 4 to 6 Mm. wide, may be of service. The knife is introduced in the same manner as the needle, and immediately following it in the next lower interarcual space, or the operation may be done a few days after the lumbar puncture.

Trephining of the skull and simple splitting of the dura mater, or puncture of the ventricles, must also be considered. Recovery has been attained in some cases of serous meningitis following otitis media by puncture of the ventricles (v. Beck and others).

## HYDROCEPHALUS

Hydrocephalus is an accumulation of fluid in the cerebral ventricles and in the lymph spaces of the brain which, as a rule, leads to ventricular dilatation. In by far the majority of cases we have to do with a hydrocephalus internus; at other times there is an effusion into the subarachnoid space, a hydrocephalus externus. A further differentiation is made between congenital and acquired hydrocephalus, but while this distinction is possible from an anatomical standpoint it is impracticable clinically, since we can never determine whether a child who develops a gradually increasing hydrocephalus in the first

years of life did not have a small hydrocephalic effusion very soon after birth, therefore congenital. The intrauterine development of congenital hydrocephalus occurs some time following the formation of the bones of the skull, therefore after the seventh fetal month (Huguenin). The causes of hydrocephalus are varied. They may be sought within or without the chambers of the brain. The extra-ventricular sources depend upon circulatory disturbances in the venous sinuses and in the large vein of Galen and its collateral branches in which the venous return is hindered. The causes of hydrocephalus which are produced within the ventricles are of different nature. It is obvious that an inflammation of the ependyma gives rise to a decided accumulation of fluid in the ventricles. The starting-point of the effusion may be sought most frequently in an inflammation of the choroid plexus. This alone may cause an effusion in the ventricles of the brain, or it may be combined with the most diverse lesions of the cerebrum.

In how far the ependyma of the ventricles plays a rôle in the development of internal hydrocephalus is unknown. In quite a number of cases which have been described under the term "essential" hydrocephalus, in which therefore the cause was undetermined, no corroborative evidence for the pathogenesis could be found at the autopsy.

Hereditary conditions have a special influence, and among these particularly the numerous nervous affections such as epilepsy, mental disease of the parents, also organic cerebral disturbances which have been brought into relation with hydrocephalus. Tuberculosis, syphilis of the parents, alcoholism, conception while under the influence of liquor (Bourneville), parental care and sorrow, trauma or profound emotional conditions during pregnancy, are also mentioned as etiologic factors in congenital hydrocephalus. Marriage of blood relatives, as well as a disproportion in the ages of the parents are said to have a bearing on the disease. After all, we can only deduce that congenital hydrocephalus has succeeded all of the conditions just mentioned, not that they are the actual progenitors of the affection.

The pathogenesis of hydrocephalus which occurs in hereditary syphilis may be referred to very different causes. Frequently there are congenital cerebral anomalies which are associated with degenerative and dystrophic processes in the brain substance; in other cases there are specific syphilitic affections which involve the ependyma, the cerebral membranes, the walls of the vessels, or the brain substance (L. D'Astros).

Inhibitive processes and deformities of the cerebrum and even of the cerebellum are often the cause of hydrocephalus, while the remainder of the nervous system may be normal. Other deformities are often associated with these malformations of the brain, such as cleft palate, bilateral club-foot, pes equino-varus, ectopia of the testicles or of the rectum, and closure of the anus. Spina bifida is sometimes present and may be complicated with meningocele or myelo-meningocele.

Congenital hydrocephalus may develop intrauterine in consequence of deformities of the brain without any post partum symptoms which would indicate

the presence of the disease; in other cases the malady arises from disease of the fetal brain of which the cause is unknown or can only be suspected.

Thus far no phenomenon indicative of an intrauterine development of hydrocephalus has been discovered, and the pregnancy differs in no wise from the normal.

Hydrocephalus may show itself after birth in the following forms. At times there is abnormal enlargement of the head or perhaps unusual smallness (microcephalia). Again, the head may at first be of normal dimensions and enlargement occurs later. Acquired chronic hydrocephalus sometimes develops soon after birth, in other cases it is latent. It may arise while the fontanelles and sutures are open or after the skull has closed. In the newborn there are certain primary infectious diseases, particularly of the gastrointestinal tract, that have a bearing on the development of the malady. Then there arises thrombosis of the venous sinuses in the skull (Marfan), or inflammation of the ependyma of the ventricles and of the meninges, or inflammation of the choroid plexus, which are associated with a profuse exudation of serous fluid. In other cases there are nutritive and circulatory disturbances of the most varied acute and chronic diseases of the brain, or hydremia which produces an increase of the exudation of cerebrospinal fluid. Occasionally in cerebral sclerosis there is an enormous effusion of fluid (hydrocephalus ex vacuo) in the ventricles, or hydrocephalus follows a distribution of the sclerosis to the ventricular walls, with an implication of the choroid plexus. The ventricle in the hemisphere which is the seat of sclerosis is more markedly dilated than the other.

Hydrocephalus may also arise from restricted circulation of the cerebrospinal fluid due to inflammatory processes, i. e., closure of the foramen of Monro causes a dilatation of the lateral ventricle and an accumulation of fluid, closure of the aqueduct of Sylvius gives rise to stasis in the second and third ventricles, and closure of the foramen of Magendi produces an accumulation of fluid in all of the ventricles. Further, hydrocephalus may develop in the course of serous meningitis and even after cerebrospinal meningitis by inflammatory changes in the plexus or in the ependyma, or by an occlusion of the basal lymph channels brought about by inflammation. Circulatory disturbances which give origin to hydrocephalus are caused by tumors upon the side of the neck and in the mediastinum which hinder the return of the venous blood from the head, and also by tumors of the brain. The latter are referred principally to the base of the brain, to the posterior cranial fossa, and especially to the cerebellum. Hydrocephalus may further arise from compression of adjacent venous trunks or of the vena magna Galeni at the side of the tumor, or from compression of the sinus by the dura mater (L. d'Astros, Barrier).

In this form of hydrocephalus the abnormal effusion of fluid leads to decided enlargement of the skull, and the cranial sutures which have been firmly closed may be separated even in children from seven to twelve years of age (Barthez, Rilliet, Bourneville). Hydrocephalus may last for months



and even years with a sudden termination in coma or convulsions and death.

Rickets is also mentioned in the pathogenesis of hydrocephalus (Pott). There is, however, by no means a specific condition dependent upon rachitis, but in the rachitic skull, in the ununited sutures, and in the softness of the bones there is sufficient stasis of the lesser circulation, such as occurs in pulmonary and cardiac affections, in the marked paroxysms of whooping-cough, in compression of the veins of the neck by tumors, etc., to produce decided exudation in the ventricles. Therefore these conditions are complications which occur in rickets. As a rule the ventricles are not at all or only moderately dilated in rickets; a decided ventricular effusion is relatively rare. On the contrary the enlargement of the rachitic skull may usually be referred to a hypertrophy of the brain or in some cases to a thickening of the cranial bones. The open fontanelles, their late closure, is due more to an enlargement of the cerebral mass than to a hydrocephalic effusion (L. d'Astros).

Meningeal or external hydrocephalus is much more rare than ventricular effusion. The former, as a rule, is of pachymeningitic origin, being due to pachymeningitis hæmorrhagica interna between the layers of the arachnoid brought about by the transformation of a hematoma into a sac containing a more or less clear fluid, sometimes sanguinolent. In what manner the transformation of the hemorrhagic into a serous effusion occurs cannot be explained.

*Acute hydrocephalus* occurs preëminently in nurslings and in the first years of life. At the onset of the disease a clinical differentiation of acute hydrocephalus from other acute cerebral affections is often impossible.

The condition usually arises in the course of infectious diseases, especially of infectious gastrointestinal catarrh in nurslings. Restlessness, gritting of the teeth, irregularity of the pulse, uneven respiration, spasm of the glottis, strabismus, pupillary inequality, and stupor are observed in the course of the affection. Tonic and clonic convulsions suddenly appear, the fontanelles become prominent, and cerebral pulsations can be felt. Sometimes the bone separates and the circumference of the head increases. Coma gradually appears, the pupils dilate and fail to react, the glance is rigid and the patient perishes. Acute hydrocephalus may be replaced by the subacute or the chronic form.

When meningeal hemorrhage from the onset is associated with convulsions and occasional fever, and is combined with contractures in the extremities (Legendre), there may be confusion with acute hydrocephalus.

At the beginning of the disease there are no definite symptoms to indicate an acute effusion into the cerebral chambers. The condition of the fontanelles and of the sutures gives no support for the occurrence of an effusion in the course of the affection. Edema of the pia mater or of the cerebral substance may produce the same symptoms, such as headache, sopor, convulsions, and coma, as are found in serous exudation in the ventricles. Ventricular serous effusion such as is observed in tuberculous meningitis, in rickets, and also in Bright's disease, will not be considered in this connection

since in all of these affections there is but a single symptom—effusion into the ventricles—not the characteristic pathologic picture that is common to hydrocephalus.

**Symptoms.**—The most important and decisive phenomenon is enlargement of the skull. Hydrocephalus is due to an accumulation of intracranial fluid. The enlargement is more marked the earlier the exudation begins, therefore when it develops intrauterine, and it may then become so great that birth is impossible unless the skull is perforated. Immediately following birth the head is more or less spherical; later, after some of the sutures have ossified and closed, while others are still capable of distension, there is a certain degree of asymmetry. As a rule the fluid is clear, light colored, and serous. In most cases there is an effusion into the cerebral ventricles; this may, however, enter the subarachnoid space and produce an external hydrocephalus. Attention is first drawn to the condition by the abnormally rapid and progressive enlargement of the skull, but these cranial changes may now and then be preceded by repeated convulsions and contractures in the muscles of the extremities. These symptoms cannot primarily be utilized in diagnosis, therefore the increasing enlargement of the skull must be the determining factor and is dependent upon the degree of cranial distension and the amount of the fluid effusion. The circumference of the skull may be 60 to 90 cm. Palpation readily demonstrates the broad sutures and enlarged fontanelles; the latter are prominent and distinctly fluctuate. The skull also shows retarded ossification and in occasional extreme cases of hydrocephalus disseminated bone nuclei may be felt in the tense and fluctuating membranes which unite the cranial bones. The enlarged head with its abnormally wide sutures, tense membranes, and thin bone, may be so transparent that the light of a burning candle, or sunlight, can be seen through the skull. The veins of the head are often dilated and the hair is scant and of slow growth. The prominence of the frontal bone is in marked contrast with the thin, triangular face. The frontal bone is arched anteriorly, the occipital bone posteriorly, the parietal and temporal bones are forced aside.

The supraorbital plate is flattened, the bulbi are often protruded and forced downward so that the white upper scleral border is visible and the lower scleral margin obscured, and about one-half of the iris is covered by the lower eyelid. In consequence of the enormous increase in volume the head cannot be held upright without support but wobbles to and fro, following the law of gravity.

Nutrition is often poor; the temperature is normal; dentition is retarded.

The nervous symptoms are of the most varied character and may arise at the onset or during the course of the affection. Often they reappear after certain intervals; their frequent recurrence as a rule indicates exacerbation of the disease. The nervous phenomena are dependent upon congestion or upon an increase of the intracranial pressure. After the pressure has become stationary and the brain has accommodated itself to this pressure all the symptoms may disappear.

At the onset of the affection there is sometimes headache, restless sleep, often there are sudden outcries. Mental development is retarded, the child for the most part is apathetic, occasionally disagreeable, does not appear to see and hear distinctly, and fails to recognize those about him. Memory suffers. Speech is incomplete, hesitating, slow, the child lalls, now and then speaks indistinct words, and not infrequently presents the picture of complete idiocy. The mental condition is not dependent so much upon the hydrocephalic effusion as upon the concomitant cerebral disturbance (Bourneville).

Movement of the upper extremities is not, as a rule, greatly affected, but there may be uncertain movements resembling chorea, particularly when the child attempts to grasp an object. In the lower extremities however there is almost always a distinct weakness and when an attempt is made to place the child on its feet the legs are crossed or raised. The gait is hindered, difficult, waddling; debility may be so great that the legs cannot support the body, or if there is a large effusion complete paralysis develops. Unilateral paralysis is extremely rare. Naturally there are also cases in which this weakness of the legs is insignificant.

From the onset of the disease until the terminal stage there are various contractions in the arms, legs, and face; spasm of the glottis, strabismus, and nystagmus are observed, and often paralytic phenomena on the part of the oculomotor and abducens nerves to which actual eclamptic attacks are added. In addition there is transitory somnolence, involuntary voidance of feces and urine in the bed, and decrease or loss of sensation. Ophthalmoscopic examination frequently reveals choked disc or pressure atrophy of the papilla nervi optici. There may be total blindness. Contractures sometimes develop in the course of chronic hydrocephalus which at first are slight. Usually the fingers are first involved, then the forearm and the lower leg, and finally the muscles of the trunk.

Hearing is not always intact, but the sensations of taste and smell undergo no demonstrable change.

Respiration, circulation and digestion may remain normal for years, but as a rule there is emaciation, the skin becomes pallid and wrinkled, the muscles are atrophic, and the child is so weak that it cannot stand, must recline in bed, and cannot support the head but rolls it from side to side upon the pillow. With the increasing weakness there are symptoms on the part of the lungs and heart such as atelectasis and broncho-pneumonia associated with cyanosis and dyspnea, or cardiac asthenia, which threaten the life of the patient.

The course is often progressive, sometimes there is sudden exacerbation of the process which is indicated by severe headache, spasm, general convulsions, and paralytic phenomena, occasionally associated with febrile symptoms, all of which are dependent upon cerebral congestive conditions.

Nervous symptoms occasionally appear even before enlargement of the skull, such as spastic muscular contractures in all of the extremities (v. Ranke, Ganghofner), and repeated convulsions have been known to precede the changes visible in the skull.



Many children die in the course of a few months or in the first years of life. Death sometimes results from an eclamptic attack; in other cases the emaciated patient succumbs from debility, or intercurrent diseases hasten the lethal outcome.

Only exceptionally is there an arrest of the process; the skull hardens and closes and the patient may reach an advanced age, but mental development is retarded. Two hydrocephalic patients, the circumference of whose skull measured 86 and 88½ cm. (L. d'Astros), attained an age respectively of 29 and 60 years.

It must, however, be remarked that exceptionally a child with a hydrocephalic skull, who in the first years of life frequently suffered from eclampsia, was retarded mentally, could scarcely lall, much less speak, and in whom there was a paralysis of the lower extremities with alternating contractures, has begun between the sixth and tenth years to develop mentally as well as physically, so that in the course of time nothing remained of the early severe symptoms. The symptom which is relatively most protracted is that of weakness in the lower extremities, but even this may disappear under the influence of a proper stimulating diet, the use of brine baths, massage, and electricity. The *prognosis*, therefore, is not absolutely hopeless throughout.

Recovery from chronic hydrocephalus may exceptionally follow spontaneous rupture of the hydrocephalic effusion through the nose, the ears, the cavity of the eye, or by traumatic influences which implicate the skull. With decrease of the effusion, and with good nutrition, the brain may develop, the bones of the skull grow stronger and fill the space of the discharged fluid, the sutures and fontanelles may close. Nevertheless this form of recovery must be considered hypothetically.

**Pathology.**—The anatomical finding reveals general enlargement of the skull and smallness of the facial bones. The head is spherical, the supra-orbital plate is flattened, the ethmoid bone depressed, the transverse diameter is greater than the sagittal measurement. The brachycephalia is more prominent the younger the individual, and is dependent upon the effusion of fluid. If the patient recovers, the peculiar shape of the skull is often noticeable in advanced age. The quantity of fluid varies from 300 to 500 c.c., and often amounts to a quart or more. Remarkably large fluid accumulations have been reported, for example in Esquirol's case, where the fluid measured 36 quarts (L. d'Astros). The composition is that of normal cerebrospinal fluid. In chronic cases it is clear and colorless, or faintly tinged with green. The reaction is alkaline, the specific gravity ranges from 1.001 to 1.009. It contains a trace of albumin, 0.5–1.3 to 1000 c.c., and in addition 5 to 8 grams of sodium chlorid. The relation of the ventricular effusion to effusion in the subarachnoid space varies. Communication of the fourth ventricle with the posterior subarachnoid space through the foramen of Magendi is not always present, especially in the first year of life (L. d'Astros). The inconstancy of this communication is of great importance from the standpoint of eventual surgical intervention.

Dilatation of the ventricle is the natural result of the effusion of fluid and shows extraordinary modifications. It may be so marked that upon removal of the top of the skull the brain protrudes like two bladders, and, after it is cut, the layers, thin as paper, permit the soft cerebral substance to collapse. The effusion is frequently distributed uniformly to both lateral ventricles, but it may be greater on one side than the other, or in one posterior horn than in the other. The foramen of Monro is enlarged, sometimes to 3 or 4 cm. in diameter. The third ventricle is dilated, sometimes also the aqueduct of Sylvius and thereby the fourth ventricle. The distension of the skull may be uniform, or premature and partial synostosis may cause more or less asymmetry. The sutures of the skull are wide, the fontanelles are uncommonly large, and the frontal fontanelle often extends to the root of the nose. The cranial bones are thin, flexible, transparent and glistening. Ossification of these bones, if it occurs at all, requires many years for its completion, partly because of the outgrowth of long serrated processes on the border of the bone, partly because of the development of new points of ossification within the sutures and fontanelles—the so-called Wormian bones (Virchow). Under the influence of the effusion the brain substance is reduced, sometimes from 2 or 3 cm. to a few millimeters, the gyri are separated, and the furrows obliterated. The brain is flattened at the base, soft, and atrophic; the cerebellum is frequently atrophic or may be altogether absent, in other cases it appears asymmetric or hypertrophied (Chiari). In marked hydrocephalus the hypertrophic cerebellum may be pressed down into the vertebral canal and resemble a cervical spina bifida, although it is in fact an encephalocele. In the walls of the ventricles, in the meninges, and in the plexus the most manifold changes occur, such as thickenings, granulations, or net-like proliferations, which are of inflammatory origin. The cerebral nerves are often atrophic, and in some few cases are absent.

The spinal cord is usually intact. Nevertheless hydromyelia of various grades has been observed (Chiari).

In acquired hydrocephalus the effusion and the disappearance of the cerebral substance are usually not so marked.

In external hydrocephalus there is pachymeningitis—an inflammatory process with extravasation of blood upon the inner surface of the dura mater which runs its course without any definite symptom-complex during life that would denote such a condition (Hench).

**Treatment.**—Chronic hydrocephalus with extensive enlargement of the skull is incurable. The administration of calomel, and layers of adhesive plaster or narrow bandages around the head to produce a uniform compression are useless. Vesicants, inunctions with tartar emetic ointment, unguentum veratrinæ, croton oil or mercurial ointment, are all ineffective. *Specific treatment* may be instituted in hydrocephalus dependent upon syphilis even though there be but a faint suspicion of infection. Nevertheless, the result of this treatment is often doubtful.

*Surgical treatment* has thus far given such poor results that upon the

whole operative interference is not often advisable. Certainly in the greater number of cases of congenital hydrocephalus we cannot expect that normal conditions for the development of the brain will be furnished by the withdrawal of fluid. In the quiescent form nothing is to be expected of surgery. In mild cases this treatment may be attempted. When the accumulation of fluid is progressive and the intracranial pressure increases, when life is threatened and functional cerebral disturbances arise, the sight suffers and severe headache appears, an operation is indicated. We must then consider *lumbar puncture* or *puncture of the skull*. Quinke's method involves the patient in least danger. In a great many cases the procedure is unsuccessful, and naturally there can be no result if no communication exists between the ventricles and the subarachnoid space. Nevertheless, an exploratory puncture should be made. Quinke inserts the needle between the third and fourth lumbar vertebræ, Chipault between the last lumbar vertebra and the sacrum. The patient is placed in the lateral posture with back bent forward and legs flexed. In a child the needle may be introduced directly between the spinous processes and conducted to a depth of 2 or 3 cm. toward the arachnoid space in the median line. The fluid is permitted to ooze slowly and only a small quantity is withdrawn at one operation. Large amounts are prohibited, since death may result from sudden relief of the brain. Therefore if spina bifida exists simultaneously the latter should not be punctured, otherwise we must expect a continual flow of cerebrospinal fluid which no known methods can control, and the patient perishes more rapidly than if puncture were not performed. After the needle is withdrawn, a dressing of iodoform collodion is applied at the point of puncture.

Exceptionally a cure has been attained by puncture (Schilling), or sometimes a transitory benefit is noted; but in most cases there is no improvement. Some authors advise direct *puncture of the ventricles* and have reported favorable results (v. Beck, Schilling, Graefe). The puncture must be performed with a fine capillary trocar and in the median line if the fontanelle is sufficiently broad, otherwise the insertion is made in its lateral angle. Only a small quantity of fluid should be allowed to escape. Puncture with succeeding injection of iodine has not been reported favorably except by Tournesco, and must at present be rejected (Monti, Ranke and L. d'Astros).

The dangers of ventricular puncture and its subsequent drainage are very great and the majority of patients perish in a few hours.

Somma advises direct sunlight upon the head daily for thirty to fifty minutes. If the patient is mentally deficient treatment must be continued in a suitable institution.

Acute hydrocephalus requires a purely symptomatic treatment suited to the underlying affection.

[Chronic hydrocephalus depending on rachitis of the cranium—craniotabes—is not so desperate. Many cases get well. It appears that the effusion is absorbed and brain substance may take its place. Big-headed rachitic children are frequently the best scholars in after life.—EDITOR.]



# INFANTILE SPINAL AND CEREBRAL PARALYSES

BY A. HOFFA, WÜRZBURG

WHILE the general picture of infantile spinal paralysis is well known and familiar to physicians, this is not the case with infantile cerebral paralysis, for we have gained our minute knowledge of the latter condition only in the last decades. Still generally unknown is the fact that cerebral paralysis is very amenable to treatment, and that quite remarkable results may be attained in cases which were formerly regarded as incurable. Further, the pathologic picture of each is often confused with the other in that spastic paralysis has been considered a special form of spinal paralysis.

A sketch of both diseases is therefore indicated, and later a description of the differential diagnosis. Finally, a discussion of their treatment will reveal what may be accomplished by a proper therapy.

## INFANTILE SPINAL PARALYSIS

In the year 1840 S. v. Heine directed the attention of the profession to a definite, well-characterized, and common form of paralysis in children, to which he gave the name infantile essential paralysis. Later (1860) he concluded that this paralysis was a disease of the spinal cord. The first actual confirmation of this theory was attained only recently by Prévost and Vulpian, Charcot, Goffroy, Roger, Money, Kussmaul, Strümpell, and others, so that now, instead of the word "essential," we quite properly use the term *infantile spinal paralysis*.

The disease occurs almost exclusively in children between the first and fourth years. According to my statistics the proportion is 1.3 to every 1000 surgical cases; in every 1000 deformities 67 are unquestioned spinal paralysis.

**Pathology.**—The *anatomical origin* of the disease is an acute inflammation which implicates a definite area of the *anterior gray substance* of the spinal cord. The usual lesion which is found at autopsy in cases of long standing is a decided *atrophy of the anterior horn*, which is changed into coarse sclerotic tissue, often permeated by dilated and thickened vessels, and which contains scarcely any normal ganglion cells. From the primary focus of the disease a secondary *degeneration* develops and distributes itself toward the periphery, implicating the corresponding *anterior roots*, the *motor nerves* belonging to

them, and the muscles supplied by the latter. Therefore, in the paralyzed muscles and nerves we find a high-graded *degenerative atrophy*.

**Symptoms.**—*The disease almost always begins suddenly* and attacks previously entirely healthy and sound children. The initial symptoms are high fever, headache, pain in the back and in the limbs, mental confusion or complete loss of consciousness, accompanied by local or even general convulsions, all of which usually disappear rapidly. After the conclusion of this first stage there is a more or less well-distributed *paralysis*. Both legs, or the legs and an arm, or all four extremities, and perhaps also the muscles of the trunk are implicated. *This extensive paralysis is not persistent*. On the contrary, it shows rapid regression and localizes itself in that region which is permanently paralyzed. In 7 out of every 10 cases only *one* limb is involved, usually the left leg; next in order of frequency is paralysis of both legs. Paralysis of all the extremities or of both arms, as well as simple and crossed hemiplegias, are rare. In the great majority of cases the extensors are paralyzed.

While the general condition of the children improves, the paralysis which remains is exclusively *flaccid and atrophic*. A few weeks after the onset of the malady the affected muscles show distinct *atrophy* which gradually increases to an extreme degree. Occasionally it is concealed by a *marked development of fatty tissue*. Even more rapid than the apparent atrophy are the *changes in electric contractility* of the paralyzed muscles. There is a well-developed *reaction of degeneration* (DeR). Very frequently the growth of the affected extremity is retarded so that later the *bones show a shortening of several centimeters*. However, as was shown by Volkmann, there is not always a parallelism between the muscular atrophy and the inhibited growth. The *tendon and cutaneous reflexes* are almost always absent. *Cutaneous trophic disturbances* are common; the skin is cool and often has a cyanotic appearance; *sensation, however, is completely retained*. Bladder and rectum are seldom paralyzed. A further result of this muscle and nerve paralysis is the *paralytic contracture*, to which we must now direct our attention.

For the proper understanding of these paralytic contractures a physiologic description of the antagonism of muscles is absolutely necessary. Following Riedel, this may be best accomplished by study of a normal muscle, the tendon of which has been severed.

When a tendon is cut or is separated from its osseous support the elasticity of the muscle causes a permanent retraction. The muscle flies back like a piece of India rubber which has been made taut and then cut. As the intermuscular connective tissues do not contain coarse and especially elastic fibers the detached muscle may remain outstretched for some time, but eventually it contracts constantly more and more until it can no longer be extended. The muscular substance, however, for a long time remains unchanged, at least macroscopically; but it finally presents the typical picture of *myositis fibrosa*.

In the antagonists of the severed muscle the condition is different. At the moment when the tendon is separated the antagonist contracts. Simultaneously it contracts in consequence of its elasticity and remains shortened perma-

nently because it is not capable of extension *actively* beyond the measure of its relaxation; it does not, however, contract like the muscle which has been severed in its continuity, because it is capable of extension *passively* and involuntarily. If, for example, the flexor tendon of a finger is separated, the finger assumes the position of extension. This, however, will be altered involuntarily because of conjoint action in carrying out movements with the other fingers, as well as passively in the sense of finger flexion, as it is impossible to keep a permanently extended finger in constant repose. If the hand were not used at all the antagonist would atrophy from disuse. This condition affects the antagonists of the foot much more frequently than those of the hand because the action of the toes is less than of the fingers, particularly if other injury demands rest of the foot. After extensive tears of tendons on the dorsum of the foot we occasionally note marked atrophy of the flexors of the toes, so that the latter present complete distortion and fixation. Careful tenotomy of a portion of the extensor tendons does not usually produce this contraction, because the affected toe is implicated too much in the movements of the others (Riedel).

*Furthermore, the elastically retracted antagonist retains the property of contraction under the impulse of the will, and the repeated contraction and relaxation thereby occurring may prevent atrophy, although naturally the effect of the contraction cannot be very great.*

*The elastic retraction of the antagonist overcomes great resistance, even that of gravity.* This may be recognized in a transverse separation of the radial nerve high on the upper arm. Such an injury may be said to be analogous to separation of tendons, for it has the same effect on the muscle, *the action of the completely paralyzed muscle being quite similar to that of the severed tendon.*

In such an injury to the radial nerve we note at once that the hand assumes a permanent flexure immaterial whether the forearm is pronated or supinated. *The hand remains supine even in opposition to gravity.*

We may now return to the consideration of the paralyzed muscle.

From what has been stated it is evident that the *completely paralyzed muscle will shorten in consequence of its inherent elasticity.*

This elasticity is, however, by no means identical with what was formerly designated as muscle tonus. By this was understood a constant, weak, involuntary contraction of the muscle, dependent upon the nervous system, and, since Delpech's promulgation of the *antagonistic theory* up to recent times, all paralytic contractures were explained by *this tonus*, in that the non-paralyzed antagonists were able by virtue of their tonus to draw the affected extremity to their side.

This theory of muscle tonus, which did not exist physiologically, was opposed by Werner in 1851 and has been entirely rejected since the researches of Hueter and v. Volkmann. These two authors deduced their observations from infantile spinal paralysis. They referred the deformities which exist in this disease principally to *mechanical influences* by demonstrating that



particularly *the weight and the implication of the paralyzed member* come under consideration. They, however, proceeded one step too far by *entirely disregarding the contraction and elasticity of the paralyzed muscle*. The credit of reinstatement and correct explanation of these factors belongs to Seeligmüller, and subsequently to Karewski, Lorenz, and Riedel.

Following Seeligmüller we now designate this hypothesis of the development of paralytic contractures, which will now be detailed, the *antagonistic-mechanical* theory.

In all paralyses wherein some of the muscles which control a joint are exclusively or predominantly paralyzed, the impulse of the will for voluntary attempts at movement, arising in the brain, can *only reach those muscles to which the nerve conduction has remained intact*. Accordingly, *the non-paralyzed antagonists* will contract and give to the member a definite position. *The joint must remain in this position* because the paralyzed muscles have not the power to stretch the voluntarily shortened antagonists, which, however, *in consequence of their elasticity* are already permanently contracted. Each new impulse of the will must follow the same course. This may finally cause such a high-graded contraction that the contractures arising therefrom will overcome gravity. Thus, in paralysis of the flexors of the leg, the intact quadriceps femoris, in opposition to gravity, occasionally produces a genu recurvatum, while in paralysis of the plantar flexors the extensors of the foot produce a pes calcaneus.

We just remarked that the contraction "may" assume an extreme degree. The word *may* was used with intent because there are cases in which contraction does not occur notwithstanding the fact that numerous impulses of the will constantly take the same road. *In these cases, as a rule, the affected member has received much passive motion from the onset. Probably there is also a special central predisposition to contraction* which may not infrequently be recognized by a *tendency to edema* in the paralyzed part.

In any event we cannot contest the statement that *the paralytic contractures are primarily due to voluntary contraction—not the tonus!—of the non-paralyzed antagonists*. The final position, however, depends upon still other factors: the *individual gravity* of the member and its *implication* by the body weight, therefore upon *mechanical factors*. From the moment of paralysis the struggle of these agents with the unaffected muscles begins.

Which of the two is victor in this battle, or how they are united in their deleterious action, will now be investigated.

Our starting-point must be from the supposition that not a portion but *all of the muscles which control a joint are completely paralyzed*. Under such circumstances the muscles are flaccid and weak and a loose joint may develop *provided no other mechanical influence has arisen*. In the latter case, contractures may result. These can only be governed by *the conduct of mechanical forces*, and therefore will always assume the shape which is given to the member by *the gravity of its individual parts and their implication*, i. e., the position in bed or with bandages applied.

If the paralysis is *uniformly* distributed to all of the muscles of the joint, but is not *complete*, so that a portion of the muscular function is retained, the action of gravity and the use of the paralyzed limb in walking, standing, sliding, grasping, etc., will unite to produce the deformity. For the further development of contracture another factor must be considered, namely, *the greater power of the muscles of the flexor side of the joint than those of the extensor surface*. This preponderance of the flexors over the extensors is generally recognized and has been demonstrated anatomically by E. Fischer, histologically by Grützner. It is for this reason that prolonged use of the affected joint will tire the extensors more readily than the flexors, so that the latter pull the member to their side. Therefore, in this condition *flexor contractures* are usual.

If not all of the joint muscles are paralyzed, but only isolated *muscles or muscle groups* the antagonistic-mechanical theory of Seeligmüller, which was explained above, will be admirably demonstrated. The contracture is introduced by the contraction and relaxation of the antagonists. The insertions of the latter, which have been approximated, are not drawn back into their previous position, therefore their nutritive contraction follows, which, as already stated, may even overcome gravity. In general, however, the contracture is influenced by *the gravity and weight of the joint* in the following manner:

First, the gravity and weight of the joint may act in the same sense as a beginning muscle contraction. The deformity will then become permanent all the more rapidly as the power to extend the points of origin and insertion of the shortened muscles is lessened.

Consequently *the distribution of the paralytic phenomena may exclude* such a combined action; for example, the paralysis may predominantly implicate the flexors of the lower leg, and the influence of the weight of the foot may be arrested or decreased, for a time at least, by the active contraction of the extensor muscles. Nevertheless, in the majority of such cases *the external forces gain the victory over the functioning muscles* and only rarely do the latter have a decisive influence on the anomalous posture.

It must be mentioned that the shortening of the muscles, once established, especially in growing individuals, is certainly augmented still further by tropho-neurotic changes.

**Torticollis Paralytica.**—The individual paralytic deformities must now be briefly considered. Torticollis paralytica is the result of paralysis of the sternocleidomastoid muscles supplied by the spinal accessory nerve. The affection is rare; I have seen it twice only. This *paralytic wry-neck* is characterized by an abnormal position of the head, due to the preponderance of the normal muscles of the opposite side. The chin is raised and turned somewhat toward the diseased side. Voluntary rotation of the head is difficult, but may be accomplished passively. The power to turn the head is not entirely arrested, as this may be performed by uninvolved muscles.

*The absence of muscle prominence is characteristic*, provided the movements produced by the sternocleidomastoid are executed with some slight re-

sistance; for example, with the chin supported by the hand, the patient is asked to move it downward or sidewise, whereupon only the belly of the normal muscle of the sound side becomes prominent. A prolonged paralysis may cause complete contracture of the normal muscle and therefore a permanent inclination of the head.

I shall not detail the changes which occur in caput obstipum at the base of the skull, which may implicate symmetry and produce scoliotic curvature of the vertebral column.

For the positive diagnosis of torticollis paralytica Boyer advises anesthetization. During narcosis the contracture of the antagonists is overcome and the head assumes its normal position. This applies, however, only to recent cases. In affections of long standing, wherein a nutritive shortening of the antagonists has occurred, it is impossible even during anesthesia, to relieve the deformity by torsion or pressure. In the diagnosis of torticollis paralytica the presence of other paralyses is to be considered, and important data may be gleaned from the history.

**Lordosis.**—Among the *curvatures of the spine* which arise from anterior poliomyelitis we must first mention *lordosis*. This paralytic deformity may be due also to paralysis of the abdominal muscles, but particularly to paralysis of the long extensors of the back. If the abdominal muscles are paralyzed the patient is in danger of falling backward when he stands upright, as the long extensors of the back are not controlled by their antagonists upon the anterior surface of the body. The patient aids himself instinctively by causing both ileopsoas muscles to draw the lumbar portion of the spine strongly forward, and by a backward inclination of the upper trunk. Thus an extreme lordosis develops. If, on the other hand, the long extensors of the back are paralyzed, to prevent a fall forward the patient attempts to maintain his equilibrium by bending the upper trunk strongly backward. The body is then poised by the action of the abdominal muscles and its own gravity. The result again, however, is marked lordosis of the lumbo-vertebral column, for here, as the most flexible portion of the spine, the backward movement naturally occurs.

A characteristic feature of paralytic lordosis is that the curvature of the spine at once disappears when the patient is placed in the horizontal position. The vertebral column then closely approaches the horizontal plane. This form of lordosis is very rarely permanent.

Further, curvatures of the spinal column which simulate lordosis are observed in marked flexor contractures of the hip-joint; they are then the consequence of increased pelvic inclination. These cases will be considered in the description of paralytic contractures of the hip-joint.

**Paralytic scoliosis** is a more common sequel of infantile spinal paralysis, particularly if one side of the back is paralyzed. Normal muscles will usually flex the vertebral column toward their side, so that a curvature of the spine arises. However, this may occur from purely static causes. If, for instance, in a paretic affection of the muscles of the back, the trunk is to be elevated, the patient instinctively attempts to hold the spinal column, which gives him



the greatest support on account of the contact of the bones, and thereby various forms of curvature arise according to the degree of the paralysis.

Paralytic scoliosis has not until lately been properly appreciated. Thorough work has been devoted to it by Moussarrat, Hallion, Messerer, Kirmisson, Sainton and Mirallié. The reports of these investigators show that the convexity of paralytic scoliosis was directed in the majority of instances (14 times in 17 cases) to the sound side. Only thrice was the convexity of the scoliosis toward the paralyzed side.

It is characteristic of paralytic scoliosis that it becomes fixed either late or not at all, that only rarely is there pronounced curvature of the back, and that the axis rotation of the vertebra is not well marked. Consequently there is a difference in the electric contractility upon the diseased side which favors the normal muscles.

Not infrequently there is also an inhibited development of the thorax upon the paralyzed side which is responsible for a marked asymmetry of that structure. Messerer observed 8 cases of this kind, 7 of which showed the scoliotic curvature in a direction opposed to the paralysis. In one case only were paralysis and scoliosis noted upon the same side.

I have observed in my practice numerous cases of scoliosis as a sequel of infantile spinal paralysis. Here the scoliosis, which is only *secondary*, so to speak—the static form which is the sequel of the shortening of a leg or of a flexor contracture at the hip or knee—must be differentiated from actual paralytic scoliosis, i. e., *the scoliosis due to paralysis of the muscles of the back*. I had 6 cases of the latter form. In 5 of these the *convexity of the scoliosis was directed to the normal side*, which is the rule; in the remaining case the convexity was *on the paralyzed side*. I explain the formation of a curvature on the same side as this right-sided paralysis by the fact that the patient showed a constant tendency to turn toward the diseased side after he found a point of support in the bones and had a firmer hold on the borders of the vertebræ. The body, therefore, in this case was able to overcome the pulling of the sound muscles.

Passing to the **shoulder**, we must first consider the *paralytic* loose joint. The shoulder muscles, notably the deltoids and the outward rotators, are at the same time the tensors of the capsule of the shoulder-joint. Like the cartilaginous limbus in the hip-joint, they serve for the equilibrium of the arm in the shoulder. If these muscles lose their function by an anterior poliomyelitis, the tension of the capsule is relaxed, but the arm droops because of its weight, and draws the internal rotators as far downward as is permitted by the flaccid capsule. The weight of the arm plays constantly upon the atonic capsule and is thereby able to distend it considerably. This, however, destroys the function of the joint and a flail joint arises. Such a *paralysis of the shoulder* may occur without implication of the extremities.

In paralysis of the shoulder the normal curve has been lost, the acromion is prominent, below the acromion there is a deep groove which passes into a gentle convex arch in the contour of the arm. This groove has arisen from

the lowering of the arm, for the pressure of the air has forced inward the soft parts between the acromion and the head of the humerus. The arm hangs flaccidly upon the chest and is usually rotated somewhat inwardly, while the hand is in a position of marked pronation. This inward rotation of the arm with hyperpronation always occurs as soon as the action of the outward rotators has been lost, the movement of the upper arm then being subject principally to the action of the inward rotators, the pectoralis major, the latissimus dorsi and the teres major. The entire arm hangs in the flaccid capsule. The arm can be forced in the greatest excursus forward and backward, and may also be raised so high that the head of the bone reaches the plane of the joint, or may be forced so far anteriorly or posteriorly that there is at the borders an appearance of subluxation. As soon as the support of the arm is withdrawn it falls by its weight as far as the capsule permits and therefore presents a typical flail joint.

Active elevation of the upper arm is impossible, for the patient can only move the shoulder-blade, which, in consequence of flaccidity of the capsule, has no noteworthy influence upon the movement of the arm. At most partial movements of the arm may be carried out with the aid of the muscles of the chest and back. By hyperpronation of the forearm the use of the hand is decidedly limited.

The muscles not only appear by inspection to be feebly developed, but they are so in fact. Upon palpation, instead of the tense muscle we note below the skin only a soft, flaccid mass. The faradic contractility of the muscles is usually absent, while cutaneous sensation is retained.

The paralytic shoulder deformity will not disappear of itself. On the contrary, the condition increases constantly with the growth of the child. The distance of the head of the bone from the acromion, which at first may be only about 1 cm., increases more and more until finally it amounts to 3 cm. or over, and may be so great that three or four fingers can be placed in the interspace.

In addition to the deformity, the muscular atrophy, and the helplessness of almost the entire upper extremity, in the further course there is a retardation of the entire development of the affected side. The growth of bone is lessened in all of its dimensions, so that not only the upper arm but also the scapula and the clavicle are decidedly smaller in later life than those of the healthy side. The atrophy may also involve the entire upper half of the trunk.

Another consequence of infantile spinal paralysis, in so far as it affects the *shoulder*, is the *wing-like retraction of the shoulder-blades*. This deformity is due to *paralysis of the larger serratus anticus*. The affection may also be the only remaining sequel of an extensive paralysis. The diagnosis is clear, for not only is the position of the shoulder-blade perceptible, but the anterior surface of the bone may be easily palpated.

Anomalous positions of the **elbow-joint** due to muscle contractures are as infrequent as those of the shoulder. Both are opposed to contractures by the weight of the hanging limb as well as the many voluntary and involuntary movements in the extremity, which is entirely free of pain. The conditions

are somewhat different in the hand, where the comparatively weightless fingers react more readily to the traction of contracting muscles. However, even here, on account of the frequent movement of the joint, a rigid contracture is unusual and appears only late. Seeligmüller saw flexion rigidity, that is, extension contracture, 5 times. In the explanation of contractures we must always bear in mind that these will arise only when a permanent approximation of the points of insertion and origin of the muscle has occurred.

We will now describe **paralytic contracture of the fingers**—a rare sequel of infantile spinal paralysis. As a rule the paralyzed hand is flexed, while the forearm is pronated. Exceptionally different conditions may prevail, especially if there are external mechanical influences. Thus, in a patient who for years carried his hand in a sling, with the elbow-joint in a right angle, there was pure ulnar flexion. If the paralysis has affected the *radial nerve* alone, the hand hangs flaccid, can never be raised, and even dorsal flexion is impossible. If an attempt is made to voluntarily extend the flexed fingers there will be only a partial extension, which is accomplished by the action of the interossei and lumbricales, i. e., with an existing flexion of the basal phalanges only the middle and nail phalanges are extended.

If the *ulnar nerve* only is involved, the "claw-hand" develops. At the onset of the paralysis ulnar flexion and abduction of the hand and flexion of the last three fingers are arrested. Later the inhibition of the last movement leads to the characteristic partial extension contracture, with conspicuous atrophy of the soft parts in the interosseous spaces. The deformity is always most marked in the third and fourth fingers, but the other fingers are also gradually involved.

Paralysis of the *median nerve* is characterized by immovability of the second and third phalanges in the index and middle fingers, arrest of flexion, and opposition of the thumb. The flexion of the first phalanges of the four fingers is unhindered on account of the action of the interossei.

At the **hip-joint**, as in the shoulder-joint, after infantile spinal paralysis we find a *loose socket*, which has arisen from paralysis of all of the muscles surrounding the hip-joint. v. Volkmann has given a very concise description of this flaccidity: "This does not lead to a hyperextension from gradual stretching of the anterior capsular wall. Although the strongest ligament in the human body, the ligamentum Bertini, is located here, nevertheless it gradually relaxes when the patient allows the full weight of the body to exert it continuously. This is brought about by letting the pelvis sag as far as this ligament permits instead of supporting it. The region of the symphysis pubis then appears to be markedly extended and the curvature of the lumbo-vertebral column is increased anteriorly. Young children in particular assume a position which greatly resembles congenital dislocation of the hip-joint."

Loose joints of this kind I have treated 5 times. In these cases the soft parts around the joint were almost completely atrophied and the head of the



bone could be distinctly palpated anteriorly. The functional disturbance is very marked, for naturally the support of such a joint is impossible.

*Paralytic dislocations of the hip-joint* are beautiful examples of the previously described antagonistic-mechanical theory of Seeligmüller concerning the development of paralytic deformities.

Paralytic luxations of the hip-joint are found only when definite muscle groups of this articulation are paralyzed, while their antagonists are still active. When the obturators and rotators of the thigh are incapable of function, and the adductors, on the other hand, are normal, there is a parietic dislocation of the head of the femur upon the ilium which results in a paralytic ilio-femoral luxation. The conditions are then about as follows: The tugging of the intact adductors, to which the paralyzed muscles can offer no resistance, causes first an adduction contracture, and later a dilatation of the posterior capsular wall. If to this is added the weight of the pelvis, the head of the thigh must be forced backward and upward against the ilium, and thereby cause a dislocation.

Inversely, paralytic dislocation of the head of the femur will appear anteriorly under the pubis—*luxatio femoris paralytica infrapubica*—when the power of the rotators and abductors is greater than that of the adductors.

Reclus and Karewski observed the development of paralytic dislocation from the preceding contracture while the patients were yet in bed.

The symptoms of paralytic dislocation are conspicuous. There are usually other paralytic deformities in the same leg. Paralytic luxation upon the ilium presents the following picture: The dislocated leg is shorter than the other. The trochanter major of the dislocated member is high (6–7–9 cm.) above the Roser-Nélaton line. The region of the acetabulum is empty; upon rotatory movements the head of the femur is felt deep in the atrophic glutei. The affected half of the pelvis is atrophic. As a rule the adductors react better to the electric current than the other muscles of the thigh. The leg may be adducted and fixed so that spontaneous abduction and outward rotation are impossible (Reclus).

In anterior paralytic dislocation the leg is flexed, abducted and rotated outward. Movability in the direction of extension and adduction is decreased. The dislocated head of the bone may be felt alongside the descending ramus of the pubis. The trochanter is hidden under the glutei.

Reposition of paralytic luxation of long standing is impossible even under anesthesia.

These contractures become more rigid in the course of time; the capsule of the joint and the ilio-femoral ligament contract more and more and completely fix the joint in its pathologic position. As in contractures of the hip-joint after inflammation, here also there is a secondary development of lordosis of the lumbo-vertebral column. A compensatory lateral curvature of the spine also results from shortening of the paralyzed leg, due to static influences.

**In deformities of the knee-joint** which follow infantile spinal paralysis,

it must first be remarked that of the muscles of the lower extremity the quadriceps femoris alone is paretic, i. e., incompletely paralyzed—a condition by no means rare. Therefore contracture of the knee-joint does not occur, but, on the contrary, the joint becomes too motile, and always in the same manner: it is hyperextended and a *genu recurvatum* develops. The mechanism of this deformity is comprehensible. v. Volkmann has explained it convincingly by comparison with a pocket knife: "Take a pen-knife in the hand and support it upon a table by the point, the back of the knife being turned away. The blade corresponds with the lower leg, the joint with the knee, the clasp with the thigh, the hand, which holds the clasp, with the body of the patient. It is now possible, as will be at once apparent, by slight changes in the direction of pressure, to have the blade move in the joint. Everything depends upon how the power, which is represented by the pressure of the hand, maintains itself on the joint around which the blade moves. If the power falls posteriorly, i. e., upon the edge of the blade, the knife closes under too firm pressure; if it falls in front the knife opens, and when it is completely extended we may employ full power upon the handle."

Exactly as with this pocket-knife is the mechanism of walking and standing, the extensor muscles being paretic. The patient cannot actually extend his knee-joint. To place the lower leg forward he makes a pendulous movement of the extremity. If the knee-joint were flexed the patient would fall when the foot is placed on the floor because the resistance of the quadriceps is absent—just as the pocket-knife closes, in the manner described above, when the power is directed upon the blade. Therefore, to maintain equilibrium on his paralyzed leg the patient makes the body weight so act upon the knee as to bring this joint into a position of extreme extension and maintain it thus. The leg can then bend neither anteriorly nor posteriorly. Anteriorly the weight of the body presses the joint surfaces of the femur and tibia firmly together. Posteriorly the ligamentous apparatus prevents the opening of the joint. Ligaments and bones must carry the entire body weight. Under this burden the ligamentous apparatus must necessarily suffer, particularly as nutrition itself is not very good in consequence of general atrophy. Slow but constant relaxation occurs, so that the knee-joint is more and more hyperextended.

The *genu recurvatum paralyticum* has developed. This does not inhibit walking; on the contrary, it makes it possible.

*Genu recurvatum* still permits walking when there is paralysis of all of the muscles which move the knee-joint, and it is even possible for the patient to walk without crutches.

The condition is different when the quadriceps is completely paralyzed but the flexor muscles remain capable of function. Then there is flexor contraction of the knee-joint, exactly as is required by Seeligmüller's theory.

The mechanism of this contracture may be analyzed in one of my cases. After a complete paralysis of all four extremities the implication of the arms entirely disappeared; in the left lower extremity there was marked paresis;

on the right side a complete paralysis of the quadriceps. In the lower leg only the pronators were exempt.

Both knee-joints showed a flexion contracture. Flexion could be demonstrated actively and passively to the maximum extent. Extension, however, was incomplete on account of the contracture of the flexor muscle. Notwithstanding all treatment the boy collapsed more and more from increasing flexion of the hip- and knee-joints and therefore became smaller and more helpless in his movements.

Finally, both legs were flexed at the knee-joint and both feet were in a high-graded position of valgus; the trunk, inclined strongly forward, rested upon the flexed legs, while the head was thrown somewhat backward. However, this zigzag attitude was only possible when the patient supported himself by both hands upon a cane, or placed his hands upon the anterior surface of his thighs. Walking was almost inhibited. When the patient attempted to raise the lower body he collapsed at once, like an inert mass.

This collapse can be readily explained by his static posture. When the upper trunk was held vertically the weight served to increase the flexor angle of the knee-joints.

On account of the incomplete action of the quadriceps the body necessarily had to collapse similarly as the pocket-knife. If, however, the trunk was inclined so far forward that the line of gravity was in the middle point of the angle of the knee-joint the weight of the upper body neither increased nor lessened this angle and equilibrium was established. If the inclination of the body forced the line of gravity in front of the middle point of the angle of the knee-joint the weight of the trunk forced an extension of the knee-joint and equilibrium was achieved by the action of the intact flexor muscles. The patient thus balanced his upper body between two antagonistic forces, the weight of the trunk, and the action of the intact muscles and the posterior surface of the thighs, and, notwithstanding paralysis of the quadriceps, he was able to stand and to walk with flexed knee-joints.

If patients with complete paralysis of the quadriceps do not walk, but always slide around the floor, lie in bed, or move upon crutches, the flexor contraction of the knee-joints often becomes extensive.

If all the muscles of the knee-joint are paralyzed a complete *loose joint* is not infrequent.

There is another condition which I have often observed in paralytic legs wherein a marked atrophy of the quadriceps has developed. It is well known that in cases of this kind the knee wabbles and extensive lateral movements may be accomplished in the knee-joint. On the other hand I have found but little mention of the fact that these patients can frequently perform *voluntary posterior subluxation* of their tibia. I have found this in 4 cases in a most pronounced form. The great flaccidity of the capsule is probably responsible for this phenomenon.

Three varieties of **paralytic foot deformity** must be differentiated. Perhaps one or several muscle groups are intact and pull the foot in their direc-



tion, thereby producing a *pes equinus*, *club-foot*, *flat-foot*, *talipes calcaneus* or *talipes cavus*; or all muscles which control the ankle-joint may be paralytic and thus the *loose foot* develops. Therefore, in the first instance the foot is *fixed*, in the second case it is flaccid. Accordingly Schwartz and Riefel (*Rev. d'Orthop.*, 1872) designate these forms of paralytic foot deformity as *fixed* and *loose joints*.

A typical loose joint is rarely observed in the foot. If such a condition exists the weight of the foot causes an equino-varus. This analogous position is sometimes brought about by contraction of some of the bundles of the gastrocnemius which have escaped complete paralysis. If the equino-varus has persisted for some time there may be a secondary contraction of the gastrocnemius, the ends of which have permanently approximated one another. The joint is then fixed in the equinus position and we have *fixation after a loose joint*. Transitions from one variety to another are frequent.

A few authors, notably, Boeckel, differentiate another paralytic foot deformity which is the result of tendon tugging or of destruction of the bone (*pieds bots tendineux-osseux* of the French). Naturally, the tugging of the non-paralyzed muscle, that is, of its tendons, is of primary importance; only secondarily—after a long or shorter existence of the deformity—does change in the bone take place. Attempts at redressment—under anesthesia—will readily determine with which of the two forms we are dealing.

We must now consider the paralytic foot deformities individually.

*Paralytic club-foot* is very common. It is especially frequent after paralysis of the motor nerves which supply the peronei and the extensores digitorum longi et breves, therefore, when the entire muscle group is involved which supplies dorsal flexion and pronation to the foot. But club-foot may also develop after paralysis of the supinators and plantar flexors when the weight of the foot counteracts the power of the antagonists. When we enter into the symptomatology we will see that the paralytic club-foot is supinated so that the sole is turned inward and the dorsum outward, simultaneously with a distinct inward rotation and drooping of the tip of the foot. In addition there is a conspicuous curvature of the toes; the great toe particularly is bent toward the sole of the foot and dislocated under the volar surface of the second toe. There is also atrophy of the lower leg and usually a shortening of the entire extremity; clonus and a livid discoloration of the skin are noted. The history will establish the diagnosis.

The development of *paralytic pes equinus* is easily explained. If the dorsal flexors of the foot are paralyzed the foot by its own weight assumes the position of *pes equinus*. This contracture may appear likewise after paralysis of all the muscles of the calf because the weight of the foot counteracts the action of the dorsal flexors. Occasionally we note the formation of a *talipes cavus* after a *pes equinus*.

*Paralytic flat-foot* occurs usually in paralysis of the plantar flexors and supinators of the foot but it may also follow paralysis of all muscles.

While paralytic pes equinus is usual in an immotile extremity and is caused by the weight of the anterior part of the foot, paralytic abduction—and pronation-contracture of the foot—paralytic flat-foot—commonly develops when the limb is used in walking. The weight of the body overcomes the curvature of the foot and forces it into a flat position. The soft parts and bones adapt themselves to this false position but the change is very gradual, since it is opposed by the weight of the forefoot in every elevation of the limb from the floor and when the body is recumbent.

There are also numerous cases wherein the pes valgus paralyticus arises without movement of the foot. These cases are an actual attestation of Seeligmüller's antagonistic-mechanical theory. The plantar flexors and supinators of the foot are paralyzed. Each impulse of the will is only operative by means of the intact antagonists, the dorsal flexors and the abductors, and thus the foot assumes the position of abduction. Notwithstanding this very often the weight of the anterior foot predominates. Then while the entire posterior portion of the foot shows a high-graded abduction and pronation, the anterior portion assumes a flexion position so that the appearance of the member from beneath is very characteristic. In paralytics we not infrequently find one club-foot and one flat-foot.

When the extensors of the foot and of the short plantar flexors are uncomplicated, and when there is complete paralysis of the long flexors, the active tug of the extensor may overcome the weight of the anterior foot and cause a dorsal flexion, and thus the paralytic *pes calcaneus sursum flexus* develops. This dorsal flexion of the foot may occur even before the child begins to walk. The deformity is usually aggravated by walking, and may attain a very marked grade. I have had such a patient under treatment. When walking, particularly if a genu recurvatum has arisen from insufficiency of the quadriceps, a part of the heel rests upon the floor which does not belong to the walking surface but is somewhat above the sole, toward the insertion of the Achilles tendon. In so far as the extended ligaments and the greatly deformed bones permit such locomotion, at the moment when the paralyzed foot comes into support and the body swings forward upon it, the calcaneus, which can no longer be controlled by the paralyzed muscles of the calf, is displaced anteriorly.

The paralytic pes calcaneus is frequently associated with a valgus position of the foot. As the patients grow older the weight of the anterior foot gradually bends the tarsus toward the planta and we then have the *paralytic talipes cavus*.

After this brief description of infantile spinal paralysis it need only be added that we not infrequently meet with cases in which paralysis of both lower extremities is combined with paralysis of the muscles of the back. As in all paralyses which occur in anterior poliomyelitis, we are here dealing with *flaccid paralysis*, i. e., paralysis without spastic symptoms. *Spastic symptoms*, on the contrary, characterize the infantile cerebral paralyses.

## INFANTILE CEREBRAL PARALYSIS

Passing to infantile cerebral paralysis, this in its various forms presents a large and in certain cases an extraordinarily profitable field for surgical orthopedic therapy. These patients invariably suffer from *spastic contractures*. These contractures are characterized by the shortened muscles, which are elastic but at once retract when the tension is relaxed. This gives a sensation of "springiness" upon attempted movement. Freud has attempted in his recent excellent monograph to classify the various forms of the disease which in general belong to the group of infantile cerebral paralyses. His division seems justifiable from a pathologico-anatomical aspect; in a clinico-practical sense, however, it is not so good. From a purely practical standpoint I should like to propose the classification of the cases into *four great groups*. It is true there are transitions from one group to another, but all of the unquestioned cases which come to us for treatment may be placed in one of these great groups. This is essential, for the individual prognosis, at least in my experience, depends upon our classification of the case after a careful examination.

The *first group* comprises those cases which present *spastic* muscular contractures, especially when confined to the *lower extremities*. The upper extremities are exempt, the intelligence well retained, but usually there is strabismus. *These are the only cases which should be designated true Little's disease*. I know that the excellent description of Little also includes cases of other groups, but we must go further and separate these cases clinically. However, when the spastic affection only extends to the lower extremities, leaving the upper limbs entirely free and the intelligence intact, and when, as is the rule, there is some strabismus, these cases are clinically so characteristic that they may be easily distinguished from the other forms. Therefore, in the further description of such cases I shall refer to *typical Little's disease, or to typical cases of so-called congenital spastic rigidity* (Rupprecht).

The *second great group* embraces those affections in which *not only the lower but also the upper extremities* are attacked. Here, therefore, rigidity is general. This group is designated by me as *cases of general spasticity*. These are contractures in the legs and in the arms. Simultaneously, as a rule, we have cerebral derangements (strabismus, disturbance of speech, clouded intelligence and, not infrequently, epileptic attacks).

The *third great group* includes *athetosis*.

The *fourth group* is comprised of *cerebral hemiplegias*. These cases are also well characterized. We will now devote our attention to the individual groups.

**Group I: Little's Disease; Congenital Spastic Rigidity.**—The first accurate reports of congenital spastic rigidity were by Delpech, who demonstrated that abnormal innervation of the muscles may permanently destroy the normal position of bones. Reports were also published by Heine. The first



accurate knowledge of the disease, however, originated in England, about the middle of the preceding century, when Little described the affection as a disease *sui generis*. His name has therefore been given to the malady. Recently there have been many contributions to the subject.

Particular mention must be made of the labors of Erb, Strümpell, Rupprecht, Naef, and Feer. The most complete dissertation, however, is that of

Freud, who investigated the nature of the disease, while Lorenz and Schulthess concerned themselves with treatment.

Little's disease is not uncommon; it occurs in the proportion of 4 to every 100 cases of deformity. Naef calculates that among every one hundred hospital children there is one with this disease. Thus far I have seen 21 cases. The affection is equally common among boys and girls. It is not always recognized at birth, attention being first drawn to it perhaps when the child begins to walk, and it is usually at this period that the case is brought to the physician for treatment.

The lower extremities are always implicated, usually to an equal extent.

In children the legs are commonly *rotated inward and strongly adducted* (Figs. 13 and 14), so that they are often crossed. In adults this is less marked, and the inward rotation may be entirely lacking because the adductors usually are not implicated to the same degree as in children. *The hip- and knee-joints are slightly flexed.* The position



FIG. 13.—LITTLE'S DISEASE IN CHILD, SHOWING THE INWARD ROTATION AND ADDUCTION OF THE LEGS.

of the feet may be diverse; a bilateral true pes equinus is rare. As a rule there is a bilateral pes equino-varus, but it is relatively common to find upon one

side a pes equino-varus and upon the other a pes equino-valgus. The feet of very young children usually show a quite normal position during repose; the abnormalities appear only upon first attempts to walk and are due to the tension of the muscles of the calf. According to Schulthess a *lengthening of the quadriceps tendon, the ligamentum patellæ proprium*, which leads to a *high position of the patella* and produces a peculiar sharp appearance of right-angled flexion of the knee-joint, is a pathognomonic sign of spastic spastic rigidity. Schulthess designates this elongation as a functional change of the tendon.

The trunk is inclined forward and is rigid. All *movements are awkward*. The step is short and hasty. The raised foot slides over the floor on its tip and when planted firmly it is placed either in front of or over the other foot. The knees strike each other during walking. In well-developed cases there is a very perceptible lateral movement of the trunk and shaking of the head. Usually a cane is required, or the patients cannot walk without an attendant. Stepping is particularly difficult. The child is either backward in sitting up or does not do so at all because of its inability to properly flex the thigh upon the pelvis.

The upper extremities, as already mentioned, are usually exempt, but this rule is not absolute. If they are affected, the upper arm is usually held firmly against the trunk, the elbow-joint is flexed, and the hands are pronated and flexed toward the palm and ulna. The fingers are extended or even hyperextended.

The sternocleidomastoid, the muscles of the nape of the neck, and the



FIG. 14.—SAME CASE AS FIG. 13, POSTERIOR VIEW.

muscles of the face may also be involved in the disease. In some cases the muscles of the eye are not exempt and we find *strabismus* which differs in no way from the ordinary form, and is usually convergent, as is the general rule. The eyes are free and move unhindered; the movements are not rigid nor spasmodic. Osler found nystagmus in 4 cases, and in 2 instances he noted atrophic papillæ.

The muscles of speech are often more or less involved. The child is backward in learning to speak. According to Feer in about 14 per cent. of the cases there is a slightly hindered, jerky, *slow or dragging articulation*.

The *course of the disease* is chronic, regressive or stationary. The power of the muscles, the electric contractility, and sensation are completely retained. Only the muscle tonus is abnormally increased. The quiescent muscle does not feel tense, but as soon as active or passive movements are attempted it at once shows a *condition of tonic rigidity* and presents an uneven surface. This is most marked in the muscles of the calf, the adductors of the thigh, and in the flexors of the lower leg. The antagonists of these muscles are also usually hindered in their movements. The affected muscles are commonly well developed. Their power is not decidedly diminished; voluntary movements may very readily be made if the attention of the patient is diverted. According to Adams the structure of the muscle shows little change. With insufficient exercise or entire disuse of the muscle atrophy will gradually appear, but it only becomes decided when for years the extremity has not been moved or exercised. In time actual shortenings of the most tense muscles may occur which give rise to permanent contracture; the most obvious changes are flexion and adduction of the hip-joint, strong flexion of the knee-joint, and the development of a pes equinus, club-foot or equino-varus.

Besides rigidity of the muscles there is *increased tendon reflex* which is a most noteworthy symptom of spastic rigidity. The intelligence is more or less intact; in mild cases it shows no disturbance, in others, however, there are all possible degrees of dulness up to complete idiocy. One of my patients frequently had maniacal attacks. Among our cases we find those with a high grade of intelligence, and again those in whom this function has suffered; we must be very careful not to be misled by the facial expression or by some error in speech which is so common in this disease, and to conclude therefrom a deficiency of intelligence which in fact does not exist. Convulsions appear early, usually in the first months, and almost always during the first year; they recur periodically, then grow less frequent, and may finally disappear, but sometimes they continue throughout life.

ETIOLOGY.—Little has arrived at the conclusion, based upon the observation of a large number of cases, that the cause of this disease is to be referred in almost every instance to a premature, difficult, or asphyxiated birth—a view which has been confirmed by later investigators. Feer found that the cases without cerebral symptoms may be the result of premature delivery. On the other hand, the cases with brain symptoms are to be referred to hard



or asphyxiated labor. In my patients also the history of a premature or difficult labor was found. Narrow pelvis of the mother, prolapse of the cord, primipara, etc., are sometimes causal factors.

The appearance of the disease in several children of the same family is frequently mentioned. Most noteworthy in this respect are the three children in one family, treated by Schultze. One of my patients had a brother suffering from the same disease. All of the other children were quite normal; their birth was spontaneous, while that of both the patients was artificial. There is no case on record in which heredity has been established.

**PATHOLOGIC ANATOMY.**—Difficult labor, mentioned above, gives rise to *traumatic meningeal hemorrhages* which are the principal etiologic factors in Little's disease. According to Virchow these hemorrhages result from tearing of the veins in the subarachnoid tissue where they empty from the pia into the large cerebral sinus, and are caused by the forcing together of the parietal bones. A further etiologic factor is intracerebral hemorrhage from rupture, thrombosis, or embolism. In how far inflammatory processes are concerned is yet to be determined. In any event inhibited development of the brain and disturbances of the pyramidal columns must be considered.

How such disturbances produce spastic muscular contracture may be demonstrated as follows: The motor conduction from the brain to the muscle is brought about by two neurons. One neuron proceeds from the cortex of the brain through the pyramidal tract to the ganglion cells in the gray anterior horns, and the other from here to the muscle.

Motion of the muscle is possible through the peripheral neuron alone; in fact every reflex takes place, in that the stimulation of the sensory fiber of the peripheral neuron passes to the anterior horn and from here through the motor fiber to the muscle. The reflex is an involuntary movement of the muscle. The will comes into play only as a regulating factor through the action of the central neuron upon the periphery, i. e., it inhibits the reflex movement.

Therefore, whenever the cortical motor neuron is damaged the reflex movements must be increased. In our case we *cannot be dealing with a complete interruption* of the tract which extends from the cerebral cortex to the anterior horns. Were this the case every voluntary movement of the stiffened muscles would have to be arrested. But we have already found that the rigid muscles possess the faculty of voluntary movement, therefore have not lost it entirely; that, on the contrary, the patients are able to produce movement in their joints, although at first great energy may be required. From this it must be assumed that the disturbance which we find in our patients cannot be referred to an interruption in the course of the cortical motor neuron but only to its decreased action, therefore to a disturbance in function. This fact is exceedingly important; it shows how we are to conduct our treatment. *With all the remedies at our command we must attempt to raise the energy of the cortical motor neuron and to decrease the effect of the peripheral neuron.*

**PROGNOSIS.**—Complete recovery is very doubtful. Spontaneous improve-

ment has been observed. Actual aggravation is rare; in the worst forms the affection remains stationary. Those cases are most favorable, although few, in which there is but slight implication of the muscles which pass from the pelvis to the upper and lower leg. If contractures, marked shortening of the muscles, and atrophy have already appeared a favorable result can be attained only by determined effort.

**Group II: Cases of General Spasticity.**—This group is characterized by spastic contracture of the arms as well as of the legs. In addition there are, as a rule, deficient intelligence, disturbances of speech and of sight, and occasionally epileptic attacks. These unfortunate patients we cannot aid much by any form of treatment. In one case upon which I operated death ensued, and I must therefore caution against operative interference in such patients.

**Group III: Athetosis.**<sup>1</sup>—The symptom-picture which we designate athetosis comprises the third group. The etiology of this disease is obscure, but the insignificant contractures, the *paretic symptoms* and the *spontaneous movements* are its distinguishing features. Little can be said of the pathologic anatomy. Strümpell thinks it likely that there is always a cerebral (perhaps cortical) disturbance, but the autopsy findings have not yet fully substantiated this view. The disease commonly attacks the upper extremities and preferably the terminal members, which show *almost constant, involuntary, irregular movements* of varying intensity. The legs are usually less implicated. The gait is, as a rule, spastic-ataxic in consequence of the common combination of athetosis with spastic paraplegia. There are tic-like spasms of the facial muscles which may increase to restless activity. Disturbances of intelligence and speech are more or less marked. The general clinical picture is so distinct that after one such case has been observed diagnostic error is scarcely possible.

**Group IV: Cerebral Hemiplegia.**—In the fourth group of infantile spastic contractures I have classed *cerebral hemiplegia*.

Although in this disease there is a similar pathologico-anatomical change as in congenital spasticity, nevertheless the clinical picture is so characteristic that cerebral hemiplegia may be quite properly differentiated as a special form.

All authors agree that the affection commonly arises in the first three years of life and is rare after this period. Although congenital hemiplegic cerebral paralyses are not unknown, nevertheless the overwhelming majority of patients acquire the affection extrauterine. According to Arend almost one-third of the cases may be referred to some infectious disease (measles, scarlet fever, particularly syphilis). In one-half of the patients an etiologic factor cannot be found, and the remaining cases are referred to fright and to trauma of the head. According to a large number of writers (Heine, Richardière, Sachs) *heredity* also plays a rôle.

The child, who previously has been well, is attacked suddenly with severe or with less marked initial symptoms (*vomiting, fever, convulsions*) which

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<sup>1</sup> See volume on "Diseases of the Nervous System," p. 899.

are succeeded by a *hemiplegic paralysis* which, as the name suggests, *affects one side of the body*. Combined with this there are often *aphasia* and disturbances of speech and intelligence. The frequent appearance of post-hemiplegic chorea and epilepsy renders the prognosis grave. The paralysis is referred particularly to the arm and leg; less often the face is implicated.

The upper extremity is commonly attacked more severely than the lower, and improves less rapidly. The reflexes are usually increased. On the other hand, sensory disturbances are rare. At first the paralysis is flaccid; only gradually does contracture appear, which, however, is never very marked.

*The position of the paralyzed extremities is particularly characteristic.* The arm is pressed against the trunk, the forearm is in half pronation and is flexed at a right angle with the upper arm. The elbow rests against the body. The hand is flexed toward the ulna, the fingers bent more or less decidedly toward the palm, covering the thumb (Fig. 15). The leg is rotated slightly inward and sometimes shows moderate flexion of the lower toward the upper leg with extension of the foot. The tip of the foot is turned inward, and suggests an equino-varus. In the majority of patients the great toe is raised at a right angle to the metatarsus.

The diagnosis of hemiplegia may usually be made at once. If the child is seated upon a level the arm assumes the characteristic position above described, and the knee of the same side will not rest upon the surface.



FIG. 15.—CEREBRAL HEMIPLEGIA.

## DIFFERENTIAL DIAGNOSIS

A few words must now be devoted to the differential diagnosis of infantile spinal and cerebral paralysis. This is best done by a contrast of the principal symptoms.

### Infantile *Spinal* Paralysis.

As a rule suddenly, after a febrile disease, in an otherwise normal child.

#### ORIGIN

#### ONSET

Usually without spasmodic symptoms.

### Infantile *Cerebral* Paralysis.

Often premature birth, or severe labor, with marked asphyxia.

Frequently with convulsions.



## NATURE AND DISTRIBUTION OF THE PARALYSIS

All of the extremities are commonly attacked; later the paralysis subsides in most of the muscles and is thereafter limited to one extremity or muscle group.

The paralysis is flaccid.

The paralysis affects both lower extremities, or is frequently unilateral; very often there is a simultaneous facial paralysis. The paralysis is spastic.

## REFLEXES

Usually absent.

Always increased.

## ELECTRIC REACTION

Frequent De R.

Faradic and galvanic normal.

## MENTAL DEVELOPMENT

Normal.

Often deficient.

## EYES

Normal.

Very frequently strabismus.

A discussion of infantile spinal paralysis as contrasted with other similar diseases would lead us too far; I shall only remark that the differentiation of spinal paralysis from progressive muscular atrophy, acute transverse myelitis, cerebrospinal meningitis, diphtheritic and pseudohypertrophic paralysis is to be considered.

## PROGNOSIS

The prognosis of both paralyses has already been indicated. A detailed discussion is unnecessary; as has already been stated, the prognosis of both affections, thanks to the advance of orthopedic surgery, has improved in the last decades to an extraordinary extent. Even in the severest cases of infantile spinal and cerebral paralysis we are to-day able to attain surprising results and to give returned freedom of movement to children who in earlier times would have been permanently crippled.

## TREATMENT

It is impossible for me to detail the *treatment* of the individual forms of paralysis. For this I must refer the reader to text-books on orthopedic surgery. On the other hand, it is quite easy to sketch the general fundamental principles under which the treatment is to be conducted.

We will first consider the *general treatment of paralytic contractures*, which is to be *instituted as early as possible*. Paralysis resulting from anterior poliomyelitis shows in its first stage, as we have seen, a great tendency to spontaneous cure. This is evinced by the fact that, as a rule, the paralysis of the limbs originally affected rapidly decreases and limits itself to a definite group of muscles. But the paralysis, with its sequelæ, should not be permitted to become permanent. From the beginning we should assist it in its attempts at self-cure. Therefore, the child attacked by poliomyelitis should be treated from the onset of the paralysis by methods directed against rigidity of the muscle and to prevent paralytic contractures.

The administration of internal medicaments such as strychnin or ergotin,

so recently in common use among physicians, is not advisable. These remedies may be prescribed, but local treatment of the paralysis must be instituted from the beginning. This is and will remain the principal factor in the therapy of these diseases. Therefore, at the onset of the malady there are two indications to fulfill: First, to combat the paralysis, second, to prevent the development of paralytic contractures.

The greatest success in bringing about function of the paralyzed muscle is offered by the long-continued *electric treatment* of the muscles by *galvanization*. A large, broad electrode, used as the anode, is placed upon the vertebral column at a point corresponding to the lesion in the spinal cord—therefore, in paralysis of the upper extremities, upon the cervical vertebræ; in paralysis of the leg, upon the thoracic vertebræ; in paralysis of the bladder and rectum, upon the lumbar vertebræ—while the other electrode, used as a cathode, is placed peripherally over the paralyzed nerves and muscles, or the individual muscles are stimulated by a stable current or by Voltaic alternatives. Quite powerful currents must be used. These are applied for two or three minutes and are concluded by a few changes in the current to produce contraction of the muscle. This method of electrical application is painful and can be accomplished only under energetic treatment. The parents of the child should not be allowed to make the applications. If no contraction of the muscle is produced we alternate the constant with the faradic current, one day using the constant and upon the succeeding day the faradic. Occasionally tenotomy of the contracted muscles is necessary. This relieves the tugging of the shortened muscle and lengthens the tendons by allowing the paretic antagonists more space and freer movement. The antagonists then offer little or no resistance, so that the muscles contract more readily under the current (Billroth).

The electrical applications should be followed by *massage, gymnastics, and redressment manipulations*. Effleurage, pétrissage, and tapotement are practised upon the muscles, and the joints are pushed into their normal positions. The use of these muscles, no matter how little, is advantageous. The active movements are then utilized as much as possible, and gradually opposition is exerted which includes movements of slight resistance. For paralytic contractures of the upper extremity the use of Krukenberg's automatic pendular apparatus is advised.

The parents are instructed to give the children, before they are brought to the physician, warm baths at a temperature of 26° to 28° C. (78.8° to 82.4° F.) which last ten to fifteen minutes, and after the bath to use spirits of camphor or of mustard, formic ether or alcohol. The vital energy of the paralyzed parts is decidedly stimulated by this treatment.

After electrization and mechanotherapy, the patient should be placed at once in a *suitable apparatus* to prevent contraction of the paralyzed muscles.<sup>1</sup> I strongly advise the Hessing's apparatus (Schienenhülsenapparat), for we

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<sup>1</sup> The same advice is given by Gibney.—EDITOR.

have learned to appreciate the excellent effect of this above all other mechanical appliances. With the aid of such apparatus, in combination with a supporting corset (combination paralysis apparatus), standing and walking may be accomplished even with complete paralysis of the lower extremities. The apparatus is worn constantly, but it may be removed before the morning bath and also before retiring at night, at which time the paralyzed limb should be wrapped in *warm cloths* for about an hour and then massaged. I have found *dry heat* to be of considerable value in the regeneration of muscular and cutaneous functions and particularly advise its application.

These simple measures, which must be employed rationally, often bring about a very satisfactory result, even in cases apparently desperate. At least, if we do not entirely conquer the paralysis, the children are again placed upon their feet and locomotion is possible, whereas otherwise they would have been condemned to remain cripples for life.

Even if the child does not come under treatment until *after contractures have developed*, we must still attempt to reproduce normal function of the joint by the use of electricity, mechanotherapy, baths, inunctions, and the application of dry heat. In addition, an important factor is the *actual orthopedic surgical treatment*, which endeavors to effect a complete rehabilitation of the normal form and shape of the limbs.

Redressment of paralytic contractures in general is much easier than of congenital deformities and should first be attempted by the hands alone. If this method is unsuccessful tenotomy should be performed or division of all of the soft parts which prevent the straightening of the part of the body involved. Operation upon the bone is rarely necessary. After completed redressment an apparatus is applied which permits movement of the joints. *The extinct muscular action is replaced by artificial muscles in the form of elastic bands*. Therefore, in proportion to the severity of the disease very good results are attained, and we might say that by the rational and methodic employment of all the methods at our command, the greater the paralysis and deformity the more remedial agents we possess.

Notwithstanding the treatment just described there yet remain a number of cases in which we fail to accomplish the desired result. These are the cases in which poliomyelitis has led to flail-joint or to complete paralysis of all muscles of the extremity so that they cannot function. It is true, even here locomotion is possible with the aid of suitable braces, but the patients are naturally caused much inconvenience throughout life by being confined to such apparatus. By *artificial stiffening of the joints* the legs may to a certain extent act as stilts; the operation employed for this purpose—*artificial ankylosis of paralyzed joints*—is designated *arthrodesis*, as proposed by its originator, Albert.

Arthrodesis may be indicated in the upper as well as in the lower extremity. The desired osseous ankylosis can only be brought about by a surgical opening of the joints, freshening of the joint ends, and their direct union; often, however, we must content ourselves with a fibrous adhesion. *Arthrod-*



*esis is only to be resorted to when the rational employment of all other methods of treatment has failed.*

Attempts have been made to bring about an *artificial increase in the growth of bone* (first reported by Ollier), and thus attain either an osseous arthrodesis or a lengthening of the shortened member. Under this category must be mentioned *cauterization of the diaphysis of the bone, periosteal scarification, puncture with nails, passive hyperemia, and painting with iodin*. The last two methods have shown particularly good results after arthrodesis.

*Transplantation of tendons*, a method which has lately been much discussed, must be somewhat explicitly described at this point. In an otherwise incurable muscular paralysis the tendons of normal but less important neighboring muscles are transplanted to tendons which have lost their power to function, and the activity of the normal muscles is thereby transmitted to those which are affected. Tendon transplantation is an invention of Nicoladoni, who first employed it in the cure of a pes calcaneus with paralysis of the muscles of the calf. The peroneal muscles were cut behind the malleolus, then the Achilles tendon was severed above the heel and its stump sewed to the central ends of the peroneal tendons. Adhesion took place and the result was excellent.

In the last few years Nicoladoni's operation has been frequently employed and advised. To-day not only the original operation of Nicoladoni—the transmission of function of a normal muscle to one that is paralyzed—is in use, but also a division of the function of a muscle so that its tendon is only partially sewed to the paralyzed muscle.

To bring a normal muscle into contact with one that is paralyzed three methods must be considered:

1. The tendon of a perfectly functioning muscle can be severed and attached to the tendon of the paralyzed muscle in its central stump, thereby giving new power to the affected muscle; this method is not often employed and then only when the normal muscle can be spared from its allotted work, for otherwise a complete extirpation is impossible without damaging the activity of the affected limb. As an example I may mention the transplantation of the normal flexor carpi ulnaris to the paralyzed extensor digitorum communis.

2. The second feasible method in tendon transplantation is severance of the tendon of the paralyzed muscle, its central stump being left entirely out of consideration, the peripheral end, however, being sewed to the normal muscle as much centrally as possible. For example, let us consider a paralytic pes equinus with involvement of the tibialis anticus but with retained power of the extensor digitorum communis longus. The tendon of the tibialis anticus is separated, the foot is brought into the greatest possible dorsal flexion, and the peripheral end of the tibialis anticus is sewed as far centrally as possible to the tendon of the extensor digitorum communis longus. Thus the ankle remains in dorsal flexion by the existing tension, but after adhesion of the tendon the over-contraction of the extensor digitorum com-

munis raises the peripheral part of the tibialis and thus brings about a condition resembling contraction of the tibialis.

3. The third modification of tendon transplantation is as follows: About one-half of the tendon of a completely normal muscle is separated and sewed, in the necessarily corrected position of the joint, to the tendon of the paralyzed muscle. Most frequently a portion of the Achilles tendon is employed so as to utilize the power of the muscles of the calf for the activity of the paralyzed peroneal muscles, the paralyzed tibialis anticus, or the paralyzed extensors of the toes.

Vulpus has proposed that the operation in which the whole or part of the tendon of a paralyzed muscle is sewed to the tendon of a functioning muscle be called an *ascending* transplantation; the operation in which the functioning tendon or a portion of it is sewed to a paralyzed tendon a *descending* transplantation. I suggest the term *passive* transplantation for the first form, because here an inactive, passive tendon is sewed to a normal one; for the second form the term *active* transplantation because here the tendon of an active muscle is transmitted to a paralyzed muscle. The combination of the two methods, which Vulpus designates *bilateral transplantation*, we would call *active-passive transplantation*.

A special form of tendon transplantation is the method advised by Lange—*periosteal tendon transplantation*. In this the muscle which conveys the power is not sewed *directly* to the paralyzed muscle but *to the periosteum*. Thus new muscular insertions are produced on the bone which do not exist normally and the desired function is established in a favorable manner.

For example, in paralysis of the extensor digitorum pedis a split portion of the tibialis anticus would be sewed to the dorsal side of the cuboid bone.

This method has decided advantages, particularly in that no atrophic tendon is employed in the formation of the new muscle.

Hand-in-hand with these *active*, *passive*, or *active-passive tendon transplantations* other operations which are designated *tendon shortening* or *tendon lengthening* are practised with serviceable results. According to my experience a combination of these with the foregoing methods is most profitable.

*Tendon shortening* may be effected in two ways: Either the tendon is severed with its ends approximating, and after the greatest possible stretching the severed ends are sewed together; or shortening is caused by the *formation of folds*, the tendon being run through with a strong silk thread, the ends of which are firmly fastened.

*Tendon lengthening* is accomplished, according to Bayer, by a stepwise splitting of the tendon, stretching of both ends, and the sewing of the transverse section.

This is not the place to discuss the technic of *actual tendon transplantation*. It is evident that before proceeding to this we must determine upon an *exact plan* of operation, and for this an *accurate electrical examination* of the muscles is absolutely necessary. If this procedure does not enable us

to decide whether the affected muscle is entirely or only partially paralyzed, we may determine that condition during the operation from *the color of the muscle*. The strongly functioning muscle is dark red; the paralyzed one, in consequence of fatty degeneration, yellowish white; the paretic muscle a varying rose red.

After tendon transplantation the affected member is fixed in a plaster bandage in a hyper-corrected position. After three or four days the skin sutures are removed through an opening in the cast. In four to eight weeks the cast is removed and the after-treatment by massage, gymnastics and electricity is instituted and continued for some weeks.

The result of transplantation can be noted often after a few weeks. In other cases the desired effect arises gradually. Again, we must be satisfied if only the abnormal position of the joint is overcome and a permanent correction of the deformity has been attained.

In what manner the functioning muscle is stimulated to renewed activity is a very interesting physiologic question, but has not yet been definitely decided. To a certain extent a new muscle is produced by the transplantation which, through adaptation of the cerebral cortex, gradually reaches a certain substantiveness of innervation and function. It is a curious fact that not only such muscles may be utilized for the transmission of power as are functionally closely related to the paralyzed ones, but also other muscles of quite opposed action may be employed without a poor result.

We will now turn to the *treatment of spastic contracture*, which we have learned to recognize as a symptom of *cerebral diplegia*.

We begin with the treatment of Little's disease, which was formerly known as congenital spastic rigidity.

The treatment of these cases can only be symptomatic; as already mentioned, the voluntary stimulation of the muscles is not altogether lost but only decreased, and we must therefore attempt to strengthen this by *exercises and practice*. With perseverance and rational treatment good results may be attained, and the patients will often make great progress as soon as they have overcome the first difficulties. A complete cure is naturally impossible, but we may so improve the condition that the patient can move about without foreign aid—a result which has attended all of my cases.

As we have seen, the flexors and adductors predominate over the extensors and abductors. It must therefore be our aim to reinforce the disturbed equilibrium between these opposing muscles. We must attempt to *strengthen the extensors and abductors and weaken the flexors and adductors*. The former is accomplished by massage and gymnastics. The muscles are kneaded twice daily. Systematic exercises are then instituted and continued for a long time. I begin with passive movements of the individual joints, first of the ankle, then the patient attempts active movement. After this treatment has been maintained a sufficiently long time and is easily performed by the patient, resistance movements are employed. I have my older patients practise daily with the Krukenberg pendular apparatus.



To weaken the flexors and adductors a special form of massage is employed, namely, *energetic tapotement of the affected tendon ends*. We have found empirically that this tapotement acts promptly in relieving spasm.

However, if the muscles are very rigid and marked contracture exists, tapotement is not sufficient; then, to weaken the muscles, tenotomy or tenonectomy must be performed.

By tenotomy or tenonectomy we lengthen the affected muscles and thus secure the desired release of the contracture. The adductors, the muscles of the popliteal space, and the Achilles tendon here come into consideration and it is advisable to carry out all of these operations with the patient in the sitting posture and subsequently to *encase the joints in a plaster cast in a hyper-corrected position*. Special care must be had that the legs are rotated sufficiently outward in the cast. This plaster cast is worn four to six weeks, at which time the contractures should have disappeared. But this by no means terminates the treatment; on the contrary, the principal object yet follows, i. e., the patients are taught to stand and walk by suitable gymnastics and massage. If this is overlooked we naturally will fail to have a good result. Strohmayr, who has seen only negative results from tenotomy, is certainly in error when he maintains that the operation is useless; it is only useless when no suitable after-treatment is employed.

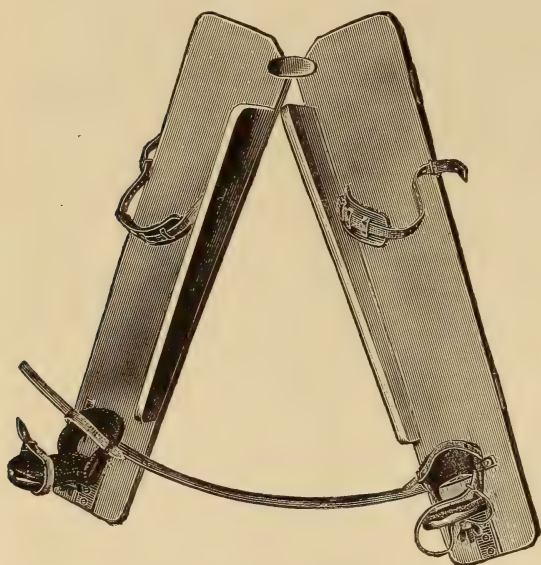


FIG. 16.—SPLINT FOR INFANTILE PARALYSIS.

Achilles tendon for transplantation upon the extensors and then lengthened the remaining part of the tendon by Bayer's method. Thus one operation enables us to elongate the Achilles tendon, to relieve the plantar flexion of the foot, and to transplant a portion of the Achilles tendon for the transmission of power to the muscles that are to be strengthened.

*Tendinoplasty* is also of great value in the treatment of spastic contractures in so far as the method of Eulenburg and Sonnenburg has demonstrated that a portion of the surplus power of the spastically contracted muscle may thereby be transmitted to its antagonist and thus weaken the spastic muscle. At the same time it strengthens the antagonist; for example, in spastic clubfoot a portion of the Achilles tendon is transferred to the peronei or to the extensors of the foot. I have repeatedly operated by first separating a portion of the

The *after-treatment* has for its purpose to maintain the *hyper-correction of the joints*. Lorenz places bags of shot upon the joints. I have employed instead a simple apparatus in which the patients are placed twice daily for two hours after massage (Fig. 16). It consists of two boards which are united by a joint which allows them to spread. The degree of the angle may be read from a scale on a steel plate which is placed at the bottom of the board. At the middle there is a leather strap which encircles the knee and holds it in position. At the lower end there is a movable foot-brace to which the feet are strapped. With this apparatus I am able to abduct the leg, to extend the knee-joint, and to rotate the limbs externally as far as is desired.

After the patients have remained in this brace a sufficient time, walking exercises in a combined paralysis apparatus are permitted (Fig. 17), first with the aid of Heusner's walking frame, then upon two sticks, and finally without any assistance. In these walking exercises it must be particularly observed that the patients plant the feet firmly and do not stamp, but assume as elastic a step as possible.

After the walking exercises gymnastics should be practised which have for their purpose the subjection of the muscles to the will of the patient. Flexion and extension of the ankle-joint, abduction and adduction of the foot, flexion and extension of the knee-joint, abduction and



FIG. 17.—COMBINED PARALYSIS APPARATUS.

outward rotation of the hip-joint, are the movements I employ in my cases, actively and passively, with and without aid. The ataxia of these patients must be overcome in the same manner as in *tabes dorsalis*, which has shown



FIG. 18.

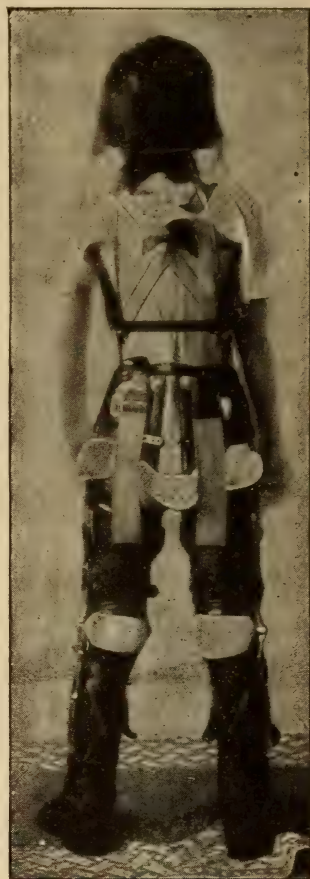


FIG. 19.

FIGS. 18 AND 19.—BRACES FOR TREATMENT OF ATHETOSIS.

excellent effects from Frenkel's compensatory reëducation treatment. In many cases I have seen beautiful results from the persistent employment of the foregoing methods.

In the *treatment of athetosis* great improvement can be obtained. Here *systematic massage, methodic exercises* and afterwards *braces* which support the head and trunk as well as both upper limbs (Figs. 18 and 19) are most valuable.

In the last few years I have attained in these cases very marked *improvement of intelligence and of the speech disturbances* by *methodic instruction in speech*.



In the treatment of *cerebral hemiplegia* the orthopedist has thus far attained but little prominence, for usually, as we have seen, the upper extremities are attacked, which as yet he is powerless to cure, and the disturbances of the lower extremities are improved without remedial agents.

However, in recent years we have been able to offer brilliant service in this realm. I have obtained the best result in affections of the lower extremity by a combined procedure. For the relief of the pes equinus I have employed tenotomy of the Achilles tendon, or Bayer's operation; then a splint-socket brace (*Schienenhülsenapparat*) is applied to which is attached a girdle for the waist, which permits an easy correction of the improper position of rotation of the extremity. In this apparatus an artificial quadriceps is produced by means of rubber bands and thereby an artificial musculature which flexes the foot dorsally. This apparatus is worn by the children for one to two years, at first constantly; later it is removed for thorough massage and gymnastics and especially for active exercises of the quadriceps and the muscles of the foot, and abduction of the hip-joint. Very good permanent results are thus attained.

*Tendon transplantation* is of great value again in this affection.

Treatment of the spastic phenomena of the upper extremity is most difficult. Here, by means of exercises, such as the Frenkel-Leyden compensatory reëducation method, such improvement might be secured as to permit the fingers in some measure to grasp again. However, the result of any treatment falls far short of complete restoration. Recently decided advances have been made by *tendinoplasty*. Here also we are now enabled to weaken the spastic muscles and to a certain extent transmit the surplus of energy to the antagonists. In several cases I have been able to reëstablish almost normal function in a hand which was previously altogether without power.

## CHOREA MINOR

BY B. BENDIX, BERLIN

SYNONYMS: *St. Vitus's Dance; Chorea St. Viti.*

The conception of chorea minor or of St. Vitus's dance (chorea St. Viti), which at various periods throughout the centuries was subject to great diversity because of the manifold modifications of the disease, is to-day controlled by a uniform and stable symptom-complex. The idea of a "*dance disease*," which the physicians of the period of the first historically positive appearance of chorea minor associated with it, is now maintained only in name (*χορεία*: dance).

The first accurate data of this "*dance disease*" dates back to 1374 when a great epidemic originated in Aix la Chapelle. At that time men and women, young and old, wandered through the city in maniacal excitement, performing fantastic dances which produced senseless rapture, and from which they did not cease until they fell down weakened and exhausted by bodily and psychical exertion. This excitement, originating principally from religious beliefs, attacked large numbers of the populace, who travelled from place to place, from land to land, so that in time the disease prevailed over entire Germany and in the countries bounding upon the north. Thus this demoniacal epidemic raged in certain regions for nearly two centuries. In the sixteenth century the affection appears to have become infrequent. About that period the conception of the disease was evidently changed, in that the people no longer resorted to unfounded and purposeless storms of dancing, but upon the eve of St. John's Day, in the chapels of St. Vitus,<sup>1</sup> they begged of their patron saint protection or cure from severe internal affections and tortures by orderly dancing which lasted several hours.

In later times, after the disappearance of the dancing epidemic, not only the dance cult was designated as chorea, but also other motiveless movements, as jumping, running, and spasmodic contortions of the body, which were frequently associated with the most varied emotional expressions as sighing, crying, laughing, etc. Those movements which we now associate with the picture of chorea were even then included. Undoubtedly, during that period two quite different affections were combined, namely, hysteria (chorea magna) and true St. Vitus's dance (chorea minor).

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<sup>1</sup> According to Witkowski, St. Vitus's dance does not obtain its name from St. Vitus, but from a very prominent Slavic god, Swantewit (St. Vit), in whose worship peculiar round dances were performed at the beginning of summer.

About the middle of the seventeenth century Sydenham, on the basis of five cases of his own observation, first clearly differentiated chorea minor from the many choreiform diseases. Notwithstanding his accurate and comprehensive description of genuine chorea and his strict limitation, we now find both names, *chorea minor* and *chorea major or magna*, side by side as *chorea anglorum* and *chorea germanorum*, without distinction from other nervous diseases, and causing frequent confusion and difference of opinion. In the year 1844 Wicke, in his "*Attempt at a Monograph of the Great St. Vitus's Dance and of the Involuntary Muscular Movements*," helped to clear the situation by giving to chorea its definite place in nosology. In this work Wicke defines Sydenham's St. Vitus's dance as "*involuntary muscular movements*." In the year 1887 Ziemssen proposed to separate chorea magna from chorea minor completely, as he saw no disease *sui generis* in the former but only "*the outflow of true psychoses and cerebral affections on the one hand, and on the other hysteria and simulation*."

This brief review of the history of St. Vitus's dance shows what manifold, varying pictures of disease were at times projected under the conception of chorea, and what changes have occurred in the course of centuries.

Therefore, I must particularly emphasize that by chorea minor we are to understand, clinically as well as *etiologically*, a well-characterized affection, *sui generis*, which has nothing in common with incurable, progressive, degenerative chorea with its peculiar movements; nor is it to be associated with the choreic movements of the heterogeneous cerebral affections. At the same time it is to be strictly differentiated from the acute paroxysms of hysteria, as well as from the lightning-like contractions of paramyoclonus (*chorea electrica*, *maladie des tics*).<sup>1</sup>

Wollenberg has proposed for genuine or Sydenham's chorea the name "*infectious chorea*." This designation, which accords to St. Vitus's dance an infectious nature or toxic origin, appears to be very acceptable, particularly upon the basis of recent etiologic observations. Heubner also favors the infectious standpoint, to which he gave expression in 1901 at the Hamburg meeting of Physicians and Naturalists in his article on chorea minor. Soltmann understands by chorea minor—St. Vitus's dance—a psychomotor neurosis of subacute course and characterized by a disturbance of coördination, by which involuntary conjoined movements are added to the movements of the will by irradiation of the will impulse, and also regards the peculiar essential idiopathic chorea of infancy as an embolic infectious disease or intoxication due to the metabolic products of pathogenic bacteria.

## SYMPTOMS

The movements of chorea minor are involuntary and are characterized principally by their interspersion between the voluntary muscular movements.

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<sup>1</sup> See volume on "Diseases of the Nervous System."



These irregular movements are particularly produced and increased by the intention of the will. They do not always bear the stamp of actual incongruity; on the contrary they are frequently the ordinary expressions of emotion and excitement, although performed involuntarily and constrainedly. We might say that they are under the influence of psychical irritation, of passion, and that occasionally they even contain an element that is somewhat theatrical.

This peculiar disease rarely begins with acute fulminant or prominent symptoms; on the contrary, it is usually of slow, gradual onset. Now and then there are prodromes, such as moderate loss of appetite and mild digestive disturbances, frequently also headache, and vertigo. The child is pale. These symptoms are not prominent and cause no concern to the parents since children who are affected by St. Vitus's dance are, as a rule, constitutionally nervous and anemic. Later, however, when the signs become more and more prominent, those about the child realize its altered mood. Previously happy, friendly, and lovable, the child now begins to show ill-humor, is disobedient, peevish, disagrees with its playfellows, and in school is inattentive and dis-trait. Illusions, fixed ideas, and loss of memory are mentioned as *rare* prodromes of chorea.

Besides these psychical changes very often there are simultaneous motor disturbances. They consist of involuntary muscular movements, especially in the trunk, pelvis, shoulders, and hands, and at first in a milder degree in the face and limbs. Thus we frequently observe a heedless play of the children with their fingers, motiveless shrugging of the shoulders, restless to-and-fro movements upon the chair. Stamping with one foot, a somewhat uncertain or dragging gait, or slight tremors in the extremities are sometimes noted.

In contrast to this subacute, gradual onset of chorea minor, very exceptionally there is a more acute appearance of the symptoms. In such cases slight fever, herpes labialis, and albuminuria may also occur.

Gradually the initial symptoms become aggravated. The slight early contractions of the face and the lightning-like movements of the eyelids and angle of the mouth are more intense and more frequent. The shoulders and the upper arm are thrust forward and inward, the fingers are seldom at rest but perform the most varied movements of extension, flexion, spreading, and grasping. The child plays with its fingers continually. The gait is spasmodic, stumbling, stamping, uncertain, waddling. The head is turned to and fro, the trunk is rotated or moved up and down. Briefly, there is a general unrest of the entire external musculature of the body. These involuntary but usually coördinated actions are not spontaneous but occur between intended movements and are therefore usually much more disagreeable to the child. The unrest and haste increase to such an extent that the child is finally incapable of performing any intended movement without interference of these involuntary coördinated movements. An exact analysis of these choreic contractions shows, as Heubner indicated years ago, that we are dealing less with movements which express impulses of the will than with movements of emo-

tion and excitement. "They are therefore complicated movements which arise in St. Vitus's dance from the pathologic irritation." As examples of these emotional contractions we frequently observe a peculiar distortion of the mouth which expresses irony, shrugging of the shoulders as a sign of indifference, or actions of anger, superiority, helplessness, or uncertainty. Soltmann, in his monograph in Gerhardt's "Handbook of Diseases of Children," which appeared in 1880, called attention to "these rapidly changing, bizarre movements, particularly of the face, which vary in intensity and extent and denote at one time pleasure and joy, at other times sorrow and care, anger or fear, and at the same time, from the rapidity of their change, usually produce a ludicrous effect."

In well-developed chorea the internal muscles also are involved in the unrest. The child has difficulty in speaking correctly, it stammers, pauses in the midst of a sentence, and, in consequence of disturbing conjoined movements of the tongue and lips, the attempts to produce an intended word may be ineffectual for a considerable time. Deglutition is often difficult. Fluids regurgitate through the nose or find their way into the larynx. Accordingly the muscles of the tongue, of the palate, and of the pharynx are affected by the disturbance in coördination. The ocular muscles sometimes participate in these movements; the eye turns hither and thither, there is transitory strabismus and diplopia. An involvement of the muscles of respiration, in particular the diaphragm, has been noted. The respiration may be irregular, at times superficial, again deep and sighing, even spasmodic. Exceptionally a hoarseness and peculiar barking cough are heard without other symptoms of disease of the respiratory organs (chorea laryngea). The heart is sometimes involved, the irregular action (arhythmic pulse) being due less perhaps to actual conjoined movements of the heart muscle (chorea cordis) than to disturbance of circulation caused by muscular spasm.

These troublesome constrained actions of chorea are most noticeable during walking; they also become particularly conspicuous in the manipulations of daily life which require either complicated movements or some firmness and repose in their performance, as in eating, buttoning clothes, putting on shoes, and writing. If the child is asked to protrude the tongue or to spread the fingers a tremor is observed instead of the usual steadiness. For writing the hand must have more than the ordinary support. The writing is characteristically changed—it becomes "scribbling."

Choreic children sometimes perform the so-called "mirror writing." This is carried out with the left hand, from right to left, and is only intelligible when the page is inverted and held to the light or reflected in a mirror. According to Soltmann, who regards mirror-writing as "the mirror of a diseased soul," this phenomenon does not belong to uncomplicated cases of chorea, but occurs in neuropathically predisposed children as an expression of cerebral disturbance.

A characteristic of the symptoms of St. Vitus's dance is that they almost always cease during sleep. Cases in which this unrest is particularly marked

at night and is absent during the day (*chorea nocturna*) are extremely rare.

The choreic movements usually are at first confined to one side and commonly in an arm; then the other arm or the leg of the same side is involved, and finally they are distributed over the entire body. In some cases the motor disturbance remains unilateral (*hemi-chorea*). In other instances the movements are bilateral, but are more prominent on one side. The preference for the left side, which is indicated by the statistics of Sée, Ziemssen and Eulenburg, is not confirmed by exact investigation, since it is found that sometimes the left, sometimes the right side, is attacked, regardless of regularity. [In the beginning the right side is more frequently affected than the left.—EDITOR.]

Gowers designates those cases of St. Vitus's dance as *chorea paralytica* in which the prominence of the choreic movements is decidedly less than the high-graded motor weakness which appears as a pseudo-paralysis of an extremity. This pseudo-paralysis or *soft chorea* ("*limp chorea*, West" or *chorea mollis*) is limited to one extremity and most frequently to the hand, or it occurs as a paraplegia or hemiplegia, or as a complete muscular relaxation of the entire body. Upon the whole chorea is rarely associated with paralysis. In addition to Gowers, Todd, West and Olivier, only Oppenheim and Nil Filatow have reported such cases. Dependent upon the time of development of the paralysis and of the choreic movements the last author differentiates three varieties of this form: The paralysis appears before the chorea and, in the course of two to six weeks is supplanted by it. The five cases of this group which were reported by Gowers were limited to a paralysis of one hand without fever, facial paralysis or pain. In the second group the paralysis occurs at the acme of the chorea; its development is rarely sudden, usually requiring several days. With an improvement of the debility either the case progresses toward complete recovery or the choreic movements are renewed. In the third series of cases the disease begins with paralysis which remains partial or becomes general, and is at no time associated with actual chorea. The diagnosis of this paralysis, as an equivalent of chorea, may be made from the absence of fever and pain, but particularly from the presence of slight digital contractions which are usually found in the paralyzed limbs of such patients. I have never observed this pseudo-paralysis in chorea, and we may assume that in isolated cases of this kind there is either an exhaustion of the muscles following the spasmodic movements, which have continued for weeks and months, or that these are cases of hysterical paralysis.

The bladder and rectum do not usually present functional disturbances. Only in one case of chorea, which was not severe, have I observed involuntary evacuation of urine and feces. The digestive functions remain normal. The urine as a rule is not altered; albuminuria is rare.

Sensory disturbances sometimes occur in addition to motor phenomena. According to my experience, cutaneous sensation remains normal in at least 95 per cent. of the cases. In the remainder there is hyperesthesia, hypesthesia, or



anesthesia. The sensitiveness to pressure of various spinous processes, mentioned by some authors, can seldom be demonstrated. It appears to be either without or in very indifferent relation to the disease itself. Reflex irritability is usually unchanged; only the patella tendon reflexes are occasionally increased.

Among the general symptoms of chorea there is loss of appetite with consequent pallor and gradual emaciation.

The psychical disturbances, already referred to in the prodromal stage, dominate the mental condition. As was then stated, the child is moody, disagreeable, readily angered, apathetic, and disinclined to play. Some patients are so extremely irritable that very slight provocation, sometimes a mere glance at them, produces the severest choreic movements which may possibly terminate in actual attacks of mania.

Sight and hearing remain absolutely intact. The reported disturbances of these senses, and perhaps also mydriasis, which has been frequently observed, are accidental conditions which have no definite causal connection with the disease.

In the severest cases of St. Vitus's dance the restlessness may become so exaggerated that all of the extremities are actually thrown about. The child can neither walk nor stand and even when kept in bed the limbs are tossed to and fro. The intensity and extent of the choreic movements may attain such a degree that the child cannot remain quiet for a moment. The tongue is bitten, swallowing is impossible, and the administration of food is extremely difficult or even impracticable. The will has lost all power over the muscles, which are uncontrollable and in such a condition of unrest as to readily disclose why the startling names "*muscle frenzy*" and "*muscle delirium*" (*folie musculaire*) have been attached to this form of chorea, instead of which Eulenburg has proposed the name "*muscle anarchy*." In these extreme cases the psychical disturbances are particularly prominent. The temperament varies constantly, the child wavers between laughing and crying, is distracted, replies incorrectly to questions, "*even his thoughts run away with him*." Sleep is restless and terminates in fright and delirium. Hallucinations arise, the depression of the mind increases to high-graded confusion which goes hand in hand with grave delirium, so that the children alarm their associates by loud outcries, such as "Fire!" "Death is here!" or "I am a thief!" True maniacal attacks are comparatively rare.

These *psychoses of chorea* which run their course under the picture of intoxication delirium, such as is observed in the acute infectious diseases, usually terminate in a comparatively brief time and only exceptionally lead to mental deterioration and idiocy.

With the appearance of acute intercurrent diseases (scarlatina, measles, pneumonia) we occasionally note an arrest of the choreic muscular movements which, however, usually reappear with greater intensity after the infectious disease has run its course. Inversely the intercurrent affection may cause a temporary aggravation of the chorea. For example, Soltmann reports a very

mild chorea in a girl aged twelve years, which appeared to run its course without any disturbance of intelligence. Suddenly, however, with the intercurrent of croupous pneumonia and increased motor disturbance, a severe maniacal delirium arose, which recurred upon three successive evenings and disappeared at the crisis.

It is an interesting fact, which I have noted repeatedly, that the relapses of chorea often terminate with the appearance of puberty.

### COURSE OF THE DISEASE

The symptoms of chorea usually require three or four weeks for their development, continue at their acme for a time, and then disappear as gradually as they developed. The total duration of the affection is not less than two, often six, and occasionally ten to twelve months. Wollenberg has calculated, on the basis of a large number of clinical cases in which the time of onset could be determined with some certainty, that the affection lasts from ten to eleven weeks. Oppenheim estimates the duration as two to three months. Now and then the so-called abortive forms (*forme fruste*) of chorea are observed, especially after infectious diseases. These rudimentary types, such as a case described by Menko which occurred after varicella, may terminate in recovery in fourteen days to four weeks. Mild cases of this kind are in contrast with chorea of protracted course, which resists cure even after a year. Cases of the latter type usually present very slight motor disturbance, with occasional exacerbations, and the patients are for the most part slender, delicate, and anemic. The permanent forms of chorea minor, which apparently are never cured, must also be mentioned, but these usually develop in later life, and we are concerned here particularly with the chorea of childhood.

The frequent tendency to *relapse* is characteristic of chorea. Sometimes one, two, or three relapses occur in the same patient; or the affection may recur every year and disappear, as above mentioned, with the advent of puberty. The attacks are not always of the same intensity. On the contrary, a severe attack may follow a mild one, or the first attack may be severe and those which follow are gradually decreased in severity and duration.

As a natural consequence of the chronic and long-protracted course a great number of cases of this type eventually show nutritive derangements—the appetite is lost, the child emaciates, loses weight, and becomes pallid.

Frequent accompaniments, or, if we prefer to call them so, *complications*, of chorea are acute *articular rheumatism* and *disease of the heart*.

Chorea occurs in the convalescent stage of rheumatism as well as in association with the disease, and both affections may arise simultaneously or the choreic movements may precede the attack of rheumatism. The conditions are similar in regard to cardiac disease. Chorea and disease of the heart, and often also the third, articular rheumatism, may appear almost simultaneously, or disease of the heart may precede the choreic affection. Again, the cardiac complication may arise as a sequel of chorea. The cardiac phenomena are

often quite mild: slight, blowing, systolic murmurs at the apex of the heart, occasionally merely an increased action, a somewhat vibrating and prominent palpable and visible apex beat with a rough character of the heart sounds. Frequently, however, a typical valvular lesion may be demonstrated, sometimes with distributed cardiac dulness, at other times with normal boundaries of the organ. In most cases of true endocarditis there is insufficiency of the mitral valve. A simultaneous stenosis is rare. Exceptionally the aortic valve may be involved or there may be an affection of the myocardium or of the pericardium. It is a noteworthy fact that with a relapse of the articular rheumatism there is usually a relapse of the chorea. We will also find that cases of chorea are particularly numerous where there is an epidemic outbreak of articular rheumatism. The frequent coincidence and the intimate relation of disease of the heart, as well as of the joint affection, with choreic movements will not be discussed at this point. Whether this coincidence is accidental or whether there is a causal connection between these pathologic processes will be more fully discussed in the etiology of the disease.

Choreic nephritis, such as has been described by Thomas and by Laache, even with a fatal termination, is extremely rare.

Hysteria has been described as an actual *sequel* of chorea, although it is very uncommon. Leube and Laache in particular report cases of St. Vitus's dance with such an outcome. Among 40 cases Laache was able to confirm this relationship twice. That genuine chorea may terminate in epilepsy appears doubtful.

### DIAGNOSIS

On the whole the diagnosis of chorea is easy. Confusion can only arise at the outset, and especially when the spasmodic movements are infrequent or only partially and feebly developed, thus conveying an impression of "awkwardness" or "wilfulness." At this period the symptoms are frequently regarded by teachers and parents only as bad behavior. Later, when the affection has progressed, very little doubt in regard to the condition of the child can remain. The characteristic, almost continuous play of the muscles, which very often involves the entire musculature of the body, the increased action in the performance of voluntary movements, the exaggeration in fixing the portion of the body affected and even upon a sharp glance at the child, the arrest of the movements during sleep, and the intact consciousness leave no doubt as to the correct diagnosis.

### PATHOLOGIC ANATOMY

The autopsy findings in chorea are of such a general nature and offer so little that is characteristic of the disease that nothing specific in regard to the pathologic process has as yet been found. The anatomic changes involve the entire central nervous system—the brain and spinal cord—and exceptionally also the peripheral nerves. In a large number of cases the necropsy findings



show positively no transformation. Thus, in 84 cases in which an autopsy was held, Sée reported 16 negative findings in the brain and spinal cord, 34 cases with changes in the heart and serous membranes, and 32 cases with softening and tuberculosis of the brain. In some cases hyperemia of the brain and spinal cord and their membranes were demonstrated *post mortem* (Soltmann, Müller). Other authors have found injection of the blood-vessels and hemorrhages into the ganglion cells (Prichard, West and Dickinson). Capillary emboli of the vessels and inflammatory changes around these, especially in the thalamus opticus and in the corpus striatum (Broadbent) have frequently been demonstrated. Meynert found nuclear proliferation in the central ganglion cells, Anton observed white cicatrices in both lenticular nuclei as the residue of inflammation, Nauwerk found widely distributed changes, partly inflammatory, partly degenerative, in the central nervous system. Eisenlohr reported a circumscribed sclerosis in the right lateral cervical cord at the autopsy of a girl aged 16 years who had suffered from congenital chorea. Elischer demonstrated principally degenerative processes in the peripheral nerve trunks. According to Ruffini the pathologic changes of chronic chorea consist in the main of a slowly progressive atrophy of the ganglion-elements of the motor cortical zone which declares itself by small calcifications, simple and pigment atrophy, disappearance of the nerve-cells, etc. These changes, when extensive, are found in the spinal cord (gray and white substances) and under some circumstances are distributed thence to the peripheral nerves and finally also to the muscular regions.

Although we are justified, as it appears, in assuming from these autopsy findings of most variable nature and localization that the cases were not always genuine chorea, but severe affections of the central nervous system with the symptomatic occurrence of choreic conditions, we may also assume that in a clinical picture so well characterized and of like appearance there are definite lesions of well localized anatomical foundation in the brain, although further investigation is necessary to fully clear up the situation.

In contrast to this inconstancy of the autopsy findings in the central nervous system the pathologic changes which are frequently noted in the heart are usually similar. In the acute cases with fatal termination delicate deposits, either recent or old, are very often found upon the valves of the heart, usually so small that they must be sought, and can be readily removed only with the moistened finger or a fine brush. These endocarditic changes occur principally in the left heart and upon the mitral valve, more rarely upon the aortic valve.

Since attempts have been made to accurately investigate these endocarditic deposits, particularly in a bacteriologic respect, very interesting facts have been demonstrated. Upon microscopic examination Naunyn found these peculiar brownish-red areas in the deposits upon the mitral valve and upon the pia mater to consist of rusty brown threads of bacteria. According to the reports of the Halle Clinic (1890), in a case of chorea the bacteriologic examination of the watery excrescences of the mitral valve as well as of the

brain showed the presence of streptococci, and in another case in which the deposits from the valves of the heart, as well as those from the spleen and from the brain, were cultivated upon agar the cultures showed a thick short rod. According to Sachs, Berkley cultivated the staphylococcus pyogenes aureus from the blood of a fatal case of chorea. Pianese obtained a bacillus from the cervical cord in a fatal case of chorea which, twenty-four hours after inoculation into dogs and rabbits under the dura, produced general convulsions or a tremor limited to certain muscle groups. The animals became apathetic, emaciated, and were exceedingly sensitive and irritable. Other authors, especially among the Italians (Maragliano, Giuzetti, and others) demonstrated staphylococci comparatively often in children who had succumbed to rheumatic chorea. In 1899 Wassermann, Westphal and Malkoff published a very interesting report concerning the bacillary foundation of chorea and its relation to articular rheumatism and to endocarditis. The reported case, in a clinical as well as a pathologic respect, bore a conspicuously infectious character. A few weeks after the onset of an acute attack of articular rheumatism the patient was attacked by choreic movements of all the muscles of the body, which rapidly increased to the severest intensity and were accompanied by paroxysmal attacks of confusion and hallucinations. The patient succumbed in severe collapse, and in addition to general hyperemia of the internal organs the autopsy revealed very fine endocarditic deposits upon the mitral valve and a recent parenchymatous nephritis. Wassermann succeeded in cultivating a microorganism from the blood of the heart, from the endocarditic deposits, from the spleen, and from the brain which, in the animal experiment, produced multiple arthritic affections. It was, however, impossible to produce by this toxin choreic movements in the animals that had been infected.

From these investigations the conclusion follows that in individuals who have succumbed to chorea the most varied bacteria (rods, streptococci, staphylococci) are found which may usually be demonstrated simultaneously in the blood and brain, in the endocarditic deposits, and in the pathologic products of articular rheumatism, and from which articular rheumatism may occasionally be produced by inoculation of animals.

## ETIOLOGY

### I. AGE

Chorea is a disease typical of childhood with a special preference for the ages between two and fifteen years. I have not observed chorea in nurslings, and the reports of such a condition by Simon, Bourson and Bouchy must be accepted with extreme caution. According to Soltmann and Heubner a physiologic congenital chorea, i. e., from birth, is impossible, as at this time an influence of the will is out of the question, therefore an irradiation of the impulse of the will cannot occur (Soltmann) and the uncomplicated, practised movements which are the expression of emotion are absent in the nursing

(Heubner). From the second to the fifth year chorea is very rare. Only after this time do we find the greater number of cases, and particularly between the seventh and fifteenth years, when the second dentition, the conclusion of the cerebral development, the marked processes of growth, and the onset of puberty, and simultaneously the deleterious influence of school life, make great demands on the youthful organism. [And both mild and marked attacks of acute inflammatory rheumatism are quite frequent.—EDITOR.]

## 2. SEX

Almost all authors who have observed a large number of cases of chorea agree that females are affected to a much greater extent than males. As a reason for this conspicuous predisposition perhaps the greater sensitiveness and irritability of the nervous system in the female is to be considered. The susceptibility of the female for chorea is about three times that of the male.

Regardless of the early statistics and figures, given in the monographs of Soltmann, Eulenburg and Wollenberg, we find in the more recent compilations the following facts:

NAME OF AUTHOR.	Number of Cases.	Males.	Females.
Koch.....	267	100—37.45%	167—62.54%
Meyer, H.....	52	20—38.46	32—61.54
Fröhlich.....	47	8—17.	39—83.
Laache.....	40	10—25.	3—75.
Brüning.....	65	15—23.	50—77.
Rabert.....	80	15—19.	65—81.

Therefore the average proportion of those who are attacked by the disease is 70–75 per cent. of girls to 25–30 per cent. boys. The wide variation in the reports may be due to a difference in the statistical material. For example, H. Meyer included only children in his compilation, while other investigators included cases which occurred in adults. Again, some of the reports are based only on dispensary practice, such as that of Eulenburg, while others combine dispensary and hospital cases. Whether coincidence plays a rôle in the variation of the figures, or whether it depends upon the greater severity of the clinical cases and hence also upon the greater susceptibility of the female sex, cannot be answered.

## 3. CLIMATE AND SEASON

Climate appears to be of secondary importance as regards the frequency of chorea. Nevertheless it must be mentioned that, according to the reports of physicians who have practised in the Antilles, St. Vitus's dance is an exceptional occurrence in that part of the world. Chorea is also rare in the tropics and is unknown in China. On the other hand it is said to be common in England and North America.



As to the influence of season the reports of individual authors do not at present permit a definite opinion. Although most writers assume a decreased frequency of chorea in the third quarter of the year, reports to the contrary are extant.

#### 4. HEREDITY

Eulenburg regards the neuropathic predisposition together with certain influences of age and special conditions in the organism, as well as a periodic increase of this abnormal predisposition, as the important predisposing factor in chorea, and Ziemssen believes that the inheritance of a markedly irritable condition of the nervous system can very often be established. Nevertheless, a careful investigation as to the hereditary neuropathic predisposition of choreic children, even within such wide limits as include with chorea various mental diseases, hysteria, migraine, nervousness, and alcoholism, finds comparatively few points of support for such views. Koch's researches among 113 cases of chorea yielded only 10 cases in which disease of the nervous system could be demonstrated in the ascendants. Chorea in the immediate ascendants has been even less frequently demonstrated. Such an occurrence must be regarded as very unusual, since Wollenberg could find chorea in the mother only 4 times in 112 cases, and Koch found the condition once in 113 cases; Rabert once in the mother and 3 times among her brothers and sisters; Brüning once among brothers and sisters; Fröhlich 3 times among the children and once in the father. Although these observations are not without interest, they by no means justify conclusions, nor do they approach the figures of other neuroses which are given as proof of the hereditary influence.

#### 5. SPECIAL PREDISPOSING CAUSES

Anemic, scrofulous and debilitated children, as well as those who have become weakened by acute or chronic diseases and nutritive disturbances, are unquestionably affected by chorea more frequently than those who are robust. Therefore anemia, chlorotic conditions, exhaustive diseases, rapid growth, improper nourishment, masturbation, the influence of school and other factors, appear to be favorable media for the development of the affection.

Chorea not uncommonly has a direct connection with acute infectious diseases. If we disregard the appearance of chorea after rheumatic affections, articular rheumatism, erythema nodosum, torticollis rheumatica, and after endocarditis and gonorrhea—their intimate affiliation with one another and with chorea has already been emphasized, and will be discussed in the pathogenesis of the disease—there are yet quite a number of infections which are occasionally succeeded by chorea. Especially do we see the affection arise after influenza, scarlatina, measles, and also after diphtheria, purulent inflammation of the tonsils, erysipelas, gastric catarrh, enteric fever, vulvo-vaginitis, etc. It may be assumed that these diseases, with the exception of the rheu-

matic affections, stand in no other relation with chorea than that they reduce the resistance of the youthful organism and thereby render it more susceptible to the "choreogenous poison."

Intense emotion, fright, anger, punishment, or shame are often the instigators of the disease (occasional predisposing cause). A direct cause cannot always be ascertained.

In the epidemics of choreic movements, such as follow the primary disease of individual children in families, schools, or pensions, and which are due to imitation and example (*chorea imitatoria*), we have not a true chorea but a hysterical affection.

Of special interest in the etiology of chorea is its connection with rheumatism. The causal relation of these two diseases was set forth by Bright and by Sée and Roger in the middle of the nineteenth century, and has since been confirmed by careful and precise investigation. Notwithstanding the fact that the relation of these two diseases has been absolutely denied by some authors, the unquestioned material at hand justifies the view that the coincidence of both diseases is causal rather than accidental. If the proof for this assumption is not evident in all of the statistics their lack of uniformity is due mainly to the fact that some authors have concerned themselves only with the question, how frequently chorea has followed an attack of articular rheumatism, and have not extended their investigations far enough to determine how often rheumatism has developed in later life upon the basis of chorea. These investigations were natural, as it is a well-known fact that chorea commonly occurs during childhood, while rheumatism is a disease of adults. [This latter assertion is modified below, as it should be.—EDITOR.] The variation in statistics also depends upon whether the rheumatic basis was limited to true articular rheumatism, or whether even vague, tearing pains have been included, as well as torticollis, erythema nodosum, erythema exudativum multiforme, gonorrhea, etc. From the recent statistics I quote the following figures: Wollenberg found in the hospital 58 per cent. and in the out-patient department 28.1 per cent. of cases of rheumatism following chorea, therefore an average of 33 per cent. Koch has found a causal relation of the two diseases only in 18.91 per cent. of his cases. Brüning determined their connection in 13.84 per cent., Heubner in 40 per cent., Fröhlich in 31.9 per cent., and Rabert in 28.75 per cent. In the Children's Clinic in Basel (Hagenbach) the material was quite different. These observations were made by Heinrich Meyer and are of special interest for the reason that they include children only, and for the further reason that Meyer traced the later course of the cases of chorea, that is, the cases that were followed by cardiac and rheumatic affections. Very interesting facts were elicited by this painstaking process, namely, that many choreic patients who previously did not show the slightest indication of rheumatic symptoms were attacked in later life by disease of the heart or by rheumatism.

In 80 per cent. of Meyer's cases a rheumatic (infectious) basis of the

chorea was established. Other causes played only a predisposing or occasional rôle. The rheumatic basis, according to this author, lies in the following facts:

(a) Chorea is the occasional equivalent for a polyarthritic attack or, after articular rheumatism, takes the place of a relapse.

(b) Simultaneously with chorea other rheumatic manifestations appear in the endocardium, in the joints, and among the general symptoms.

(c) In individuals predisposed to true rheumatism chorea may introduce the rheumatic affection.

(d) In times of the epidemic appearance of articular rheumatism chorea occurs much more frequently.

(e) Certain cases of chorea respond to antirheumatic treatment.

In addition to Meyer's investigations I must mention those interesting observations which relate to the development of chorea after diseases of the same or a very close bacterial etiology, and particularly after gonorrhea, vulvo-vaginitis, angina lacunaris, and the oft-mentioned erythema nodosum and erythema exudativum multiforme. According to these observations and well-established facts we probably have in chorea, with its peculiar and, within certain limits, always constant picture, and with its typical course, a true infectious disease. In support of this view, aside from the facts already mentioned by Meyer under (d) and (e), is the occasional introduction of chorea by prodromes and the slight rise of temperature, as in other infectious diseases, and further, the appearance of an infectious endocarditis and nephritis. The tendency of chorea to relapse reminds us of such infections as influenza, erysipelas, and pneumonia, and the preference for childhood also finds an analogon in the acute exanthemata. The autopsy reveals the evidences of an acute infectious disease: General acute hyperemia of the internal organs, frequent and very minute endocarditic deposits which are readily overlooked, as well as recent parenchymatous nephritis. Not least is the bacteriologic finding in the blood, in the joints, in the valves of the heart, and in the brain, which has been reported more and more frequently in the last decade as an infectious cause of chorea. The investigations of Westphal and Wassermann show that post-rheumatic chorea is in all probability of infectious origin, especially as both of these authors demonstrated in a positive case of chorea post-rheumatica, which terminated fatally, a streptococcus which in the animal experiment produced a multiple arthritic affection.

Therefore we will probably adopt the same conception of chorea, which Heubner has recently again set forth, and consider the etiology of the affection more from a general infectious standpoint, in that we do not look upon rheumatism as the cause of chorea but assume that "chorea is coördinated with polyarthritis and carditis and is the infantile rheumatic equivalent of the disease."



## PATHOGENESIS

From the foregoing the *pathogenesis of chorea* may be summarized as follows: Chorea belongs to the group of general infectious diseases. The affection is either due to a peculiar, definite, choreogenous poison or, more likely, may be produced by several entirely different noxæ, infections, or toxins, which individually give rise to the same clinical picture. According to the bacteriologic investigations of the last decades, the view that the bacteria or toxins which appear in chorea, endocarditis, and articular rheumatism are identical, is almost undoubted. The psychic alterations are only of predisposing or occasional moment in the development of chorea in so far as sudden fright, or the like, produces a point of lessened resistance in the nervous system, and thereby facilitates the invasion of the infectious agent, or the development and conditions of growth of a microorganism which exists normally in the body or has gained entrance thereto.

Further, in this picture and course of essential chorea, which to a certain extent is invariable, we may assume that "the toxin which has been transported to the central nervous system and slowly increases there, but which gradually becomes attenuated, exhausted or excreted," always produces the same changes in definite nervous centers. Usually the exclusive motor irritative phenomena and the psychical disturbances in the cerebrum, that is, the cortico-muscular conduction tract, may be regarded as the principal point of localization of the choreic process. That the infectious virus acts only upon a definite group of ganglion cells is a quality which it possesses in common with the poisons of tetanus and malaria, and with other specific toxins.

According to Soltmann idiopathic chorea is due also to infectious noxæ and for the most part to the poisons which give rise to rheumatism. This author regards the endocardium as the primary focus of the disease from which minute particles are loosened and transported by the circulation to the cortex of the brain, where they obstruct the tract of volition. The impulse is irradiated into improper channels and, when the innervation for movement is insufficient and does not occur at the proper time, it produces a conjoint action.

## PROGNOSIS

The prognosis of chorea may in general be regarded as favorable, but care must be taken not to calculate too short a duration. According to the last 77 cases reported from Heubner's clinic the average length of the disease is 44.3 days. The frequent relapses are also to be considered, which, however, usually cease upon the appearance of puberty. Although acute intercurrent diseases sometimes cause a sudden arrest of chorea it should be remembered that just as often a renewal of the choreic process arises after the disappearance of the acute malady. Severe complications, as affections

of the brain and pneumonia, as well as the simultaneous appearance of arthritic disease and involvement of the heart, render the prognosis more grave.

Death from chorea is exceedingly rare in childhood. Soltmann found this termination in barely 2 per cent. of the cases, and Brüning, who recently reported a compilation of 65 cases of chorea from Soltmann's Children's Hospital in Leipsic, showed a mortality of 3.1 per cent. Of the two fatal cases one was moribund at the time of admission to the hospital, so that if this case were excluded the mortality would be only 2 per cent. Death is usually not due to chorea itself but occurs mostly from complications on the part of the heart. Exceptionally acute nephritis is the cause of the fatal termination. In the severe cases death may result from exhaustion and is then usually preceded for some time by an extraordinary increase of the choreic movements together with severe psychical alteration, delirium and confusion, which finally give way to coma (Charcot's *état de mal choréique*).

### TREATMENT

In view of our conception of chorea as an infectious disease we must agree that even in the mildest cases, and especially in those of recent appearance, prolonged rest in bed is indicated. This should be continued at least until the choreic movements of the muscles decrease. If there is severe jactitation the side of the bed must be padded with pillows to protect the child from injury. Sweating should, if possible, be produced, as in the treatment of the acute infectious diseases. In the first eight to ten days of the disease, after a warm bath, or even without this procedure, the patient is completely enveloped in a wet or dry pack. After the outbreak of perspiration the child is kept in this state from one-half to one hour. If a profuse outbreak of sweat is not produced by this simple measure, even when reinforced by hot drinks such as lemonade or tea, a dose of pilocarpin (0.005–0.01) should be administered by the mouth. After sweating, the child is dried, rubbed, clothed with warm material, and left alone. If no positive success is noted after eight or ten days the treatment should be abandoned, as no results will be attained from its longer use. In some cases the quieting influence of such measures after a very short period is astonishing.

Besides rest of the body we must have mental quietude. Excitement is to be prevented, as emotional irritation has a great influence on the disposition and psychical condition of the patient. The child should be entertained, but the mind and body must not be taxed and exhausted. The mother or a good nurse is the proper companion for the child. Brothers, sisters, and playmates are to be kept away. Choreic children should not attend school; in the first place they suffer from the necessary discipline of the school, and secondly, the spasmodic movements, particularly the grimaces, expose them to teasing and ridicule and at the same time opportunity is furnished for imitation and for the development of hysterical imitative chorea

in others who are of a nervous temperament. Attempts to overcome the malady by threats or punishment are altogether wrong as chorea is not to be influenced by the will. Admittance to a hospital or similar institution I believe to be necessary only in the severe cases or where the home conditions are inadequate for proper treatment.

The diet should be non-stimulating. Spices, alcohol, tea, and coffee are forbidden. The food should be abundant, as the choreic patient, "as a working person" so to speak, has a great nutritive requirement. In severe cases, where there is difficulty in swallowing, nourishment by means of the stomach tube may be necessary.

In the further course of the disease daily outdoor exercise in good weather, or a residence in large, airy rooms is necessary.

Lukewarm baths and lukewarm ablutions of the entire body are always useful. Pick and Kraus report favorable results in the treatment of chorea from massage and passive movements gradually increased. Schilling advises treatment by suggestion.

All other treatment of chorea is by means of drugs. At the onset and particularly when the affection is combined with rheumatic symptoms and fever, the salicylates, antipyrin, analgen, exalgin, or other derivatives of the aromatic series are in place.

Favorable results are usually attained from the various preparations of arsenic, which has been employed from ancient times in the treatment of chorea. I employ Fowler's solution, in exact dosage, beginning with one drop three times a day, and after a few days gradually increasing the dose to four, six, or twelve drops, then descending in like manner to the original dose. The following mixture is given on a full stomach:

R̄	Solut. arsenical. Fowleri .....	gtt. xxx (to lx-cxx)
	Sirup. simpl. ....	30.0
	Aq. dest. ad. ....	300.0
M.	Sig.: Ten grams three times daily.	

Or in pill form:

R̄	Acid. arsenicos .....	0.01
	Mucil. gumm. arab. ....	0.5
	Pulv. rad. liquirit. ....	2.0
	M. f. pilul. No. XX.	
M.	Sig.: One or two pills three times daily.	

Nil Filatow (Klinische Vorlesungen über Kinderkrankheiten, 1901), following Comby, employs arsenious acid in the solution of 1 to 1,000:

R̄	Acid. arsenicos. ....	0.1
	Aq. dest. ....	100.0

M. Sig.: On the first day one-half teaspoonful (4-6 years) or one teaspoonful (10-12 years).



In the next seven days the dose is increased daily by the original quantity, and then decreased in the same proportion.

In the severe forms of chorea large doses of the bromids are advantageous (3-5 gm. daily):

℞ Natr. bromat.	} āā	10.0
Ammon. bromat.		
Sirup. papav.		5.0
Aq. dest. ad.....		200.0

M. Sig.: Ten grams three times daily (10-15 years).

To give the patient rest for several hours enemata of chloral hydrate are employed (1.0-1.5 per dose according to age), or sulphonal (0.3 gm. for children six years of age or 0.5 gm. for children of ten years), given at bedtime in powder form and in gruel, is a serviceable hypnotic. Morphin is not advisable.

If the child is pale and weak, iron or quinin is to be administered in combination with arsenic.

℞ Solut. arsenical. Fowleri	3.0
Tinct. quin. composit.	} āā 10.0
Tinct. ferr. pomat.	

M. Sig.: Ten to thirty drops three times a day.

If anemia is profound, iron preparations are useful, alone or in combination with quinin (Ferr. saccharat. oxydat. solub., 0.5-1 gm. three times daily, or Ferr. carb. sacch., as much as will stay on the tip of a knife, or Quin. ferro-citric., 0.1-0.2 in powder, pill, or solution).

℞ Extract. digital.	0.18
Quin. sulph.	0.3
Massæ pilul. Blandii	8.0
M. f. pilul. No. LX.	

M. Sig.: One or two pills three times daily (Soltmann).

Or

℞ Quin. sulph.	0.15
Ferr. carb. oxydat.	0.5
M. f. pulv. tal. dos. XX.	

M. Sig.: One powder three times a day.

℞ Ferr. sulph.	} āā 5.0
Kal. carbon.	
Mucil. tragac. q. s.	
M. f. pilul. No. L.	

M. Sig.: Two to five pills twice daily.

Or

℞ Ferratin	2.0-4.0
Saccharin	25.0

M. Sig.: One-half teaspoonful twice daily (Nil Filatow).

Arsenical waters containing iron (Roncegno and Levico) are serviceable if the treatment is prolonged, and especially during convalescence.

The anemia should also be influenced by the food and to that end fresh meat, meat extracts, fresh vegetables, especially spinach, as well as albumose preparations and malt are indicated.

During convalescence from this exhausting disease the debilitated and anemic children require a residence in the mountains or country, mild hydrotherapy, stimulation of the appetite, and good food.

[The best remedy for mild and severe cases is arsenic. The liquor potassii arsenitis should be given three times a day after meals, amply diluted in water. The doses should be slightly increased from day to day and may reach the double or threefold quantity; for instance  $\frac{1}{2}$  ounce may be mixed with one ounce of water. The initial dose of 6 drops may be 7 drops, 8 drops, 9 drops, etc., up to 15 or 20 or more, a drop to be added every consecutive day. If symptoms of overdosing should appear, edema of the eyelids or face, intestinal disturbance, eruptions, no increase would be allowed, or the dose slightly diminished. When no effect is attained, the dose was too small. Medicines must be given for effect, or not at all.

A dose of antipyrin with a bromid, or codein will secure a good night's rest.

When chorea persists in the night, rest must be enforced by chloral and a bromid. Bad cases must be made to sleep from 14 to 18 hours daily.

Very violent and sudden cases which are complicated with pain on pressure over the cervical part of the spine, and elevation of temperature, require ergot (and a brisk purgative, magnesium sulphate) until the symptoms of spinal congestion are quieted.—EDITOR.]

# PNEUMONIA OF CHILDREN AND ITS TREATMENT

BY E. HENOC, DRESDEN

PEDIATRICS should be the last subject of clinical study. Therefore, in describing the various forms of infantile pulmonary inflammation, which have many signs and symptoms in common with the disease in adults, I am justified in the omission of several points which I take for granted are well known, and shall merely touch upon others. I shall not present a dogmatic description, but will attempt to fulfill my object in a more clinical manner.

We will begin with an examination of two patients. The first is a boy, aged 6 years, who has been in the hospital about three days. The rapid movements of the ala nasi and, more so, the auxiliary respiratory muscles of the thorax and of the diaphragm, reveal a serious affection of the organs of respiration. There is decided fever ( $39^{\circ}$ – $40^{\circ}$  C.— $102.2^{\circ}$ – $104^{\circ}$  F.), the pulse is full and rapid (136 per minute), although not to the extent that would be expected from the increased respirations (40 per minute). The occasional dry cough produces a painful contraction of the facial muscles, and upon the cheeks there is decided flushing. No sputum can be obtained. Upon physical examination we find a normal condition of the heart, but upon the right side posteriorly from the spina scapulæ to the base, and limited by the axillary line, there is marked dulness upon percussion, and bronchial respiration and bronchophony over the same area; the remainder of the thorax presents nothing abnormal.

Examination of the second patient, a child of 3 years, who has been seriously ill for about eight days, reveals the same general symptoms—high fever, cough and dyspnea. Upon closer observation, however, it is noted that the cough is much more constant and tormenting, and the dyspnea more pronounced than in the first case. The respirations occasionally increase to 60 per minute, while the pulse is small and at the rate of 140 per minute; all of the auxiliary muscles of respiration are active, and each expiration is accompanied by a short moaning sound which is audible even during slumber. The face is pale and denotes suffering. Percussion reveals slight *bilateral* dulness at the base posteriorly, and upon auscultation there are fine, ringing râles from the middle to the base at the back which completely obliterate the normal respiratory murmur; at other areas of the thorax many râles may be heard laterally and anteriorly.

It is my purpose to describe only the principal features of the disease



and to omit the secondary phenomena, since only the former will give us a clear insight of the nature of the affection. It is obvious that in both the foregoing cases we are dealing with an *acute inflammatory affection of the lungs*, but two essentially different forms are presented. In the first patient we are concerned with a unilateral affection limited to the pulmonary tissue of the right lower lobe; in the second case there is a bilateral inflammation especially implicating the finer and finest bronchial twigs of both lower lobes. These two principal types, which are known as "*primary or fibrinous pneumonia*" and "*catarrhal or broncho-pneumonia*," will now be considered in detail. The latter disease is far more common in childhood; nevertheless, among 123 clinical cases of fibrinous pneumonia there were 26 whose ages ranged between 6 months and 3 years.

### PATHOLOGICAL ANATOMY

First a brief description of the anatomico-pathological lesions is necessary. The essential difference is in the distribution of the inflammation, which, in broncho-pneumonia, arises always in the branchings of the trachea, thence invading the finest bronchial tubes and the pulmonary alveoli; while primary or fibrinous pneumonia does not require this propagation from the bronchi but begins suddenly, implicating a large or smaller area of the parenchyma of the lung, and producing consolidation by means of a coagulable exudate. It is, therefore, evident that broncho-pneumonia is introduced by a more or less extensive catarrh; while the primary form, provided there is not an accidental preceding catarrh, begins suddenly in the midst of health. For this reason it is necessary to guard against broncho-pneumonia in children who suffer from bronchial catarrh, therefore in all infantile affections, especially measles, influenza, and whooping-cough, in which this condition is present. Fibrinous pneumonia is by no means infrequent in the course of these diseases—a fact of which I shall make mention later—but it is much less common than broncho-pneumonia, which is the most frequent cause of death in the course of these infections. This mortality is greater the *younger* the child; for it is a fact, although unexplained, that children in the first years of life show a special predisposition to this spread of the bronchial catarrh to the finest bronchial twigs, which is less evident as the age increases, so that, apart from cases of an infectious nature, the typical broncho-pneumonia which is observed daily in young children is very rare in adults, and only becomes frequent again in the aged and senile.

The anatomical form, in which both diseases appear, may be explained by this difference of development. Primary pneumonia at once attacks a large portion of the lungs—an entire lobe, or even a greater area—and transforms it, usually with great rapidity, into a compact, airless, "*hepatized*" mass, while the remainder of the lungs may be exempt. Broncho-pneumonia, however, following the course of the medium and small bronchi, distributes

itself to the pulmonary alveoli, and, primarily, to those in relation to the bronchioles already attacked by the inflammation. At first no *lobar* continuous consolidation occurs, as in the primary form, but a *lobular* one, so that only the lobules of the lung in relation to the bronchioles implicated are consolidated, in the form of coarse foci of a reddish brown or gray color, varying in size from that of peas and beans to hazel-nuts, which may be seen and felt within the parenchyma, which may still contain air. For this reason the older physicians designated this form "*lobular pneumonia*" and regarded it as characteristic of the first years of life. Very often, however, this does not terminate the condition, for the more numerous the bronchioles which are implicated in the inflammation, the more alveoli and correspondingly the more lobules are involved in the process. The hyperemic inter-spaces, which at first contain air, gradually become smaller, and the diseased lobules approximate one another and by confluence may finally lead to *extensive*, wedge-shaped consolidations which are commonly found at both lower lobes, ascending from the base. Such a condition may also be present in the upper lobes, even upon that portion of the left upper lobe adjacent to the pericardium. This extensive consolidation, designated by the French "*pneumonie lobulaire généralisée*," may give rise to perplexity in diagnosis, provided such a form of development is unfamiliar to the physician, for the physical signs show great similarity to primary pneumonia.

In addition to the differences between these two types of pneumonia which have already been described, it was formerly supposed that there were variations in the nature of the inflammatory product. It was assumed that in primary pneumonia the alveoli were filled with a solid mass consisting largely of coagulated fibrin and blood-corpuscles, while in broncho-pneumonia the contents were almost entirely fatty epithelial cells and pus-corpuscles. This view is no longer tenable. Recent investigations (Steffen, Steiner, Damaschino, Cadet de Gassicourt and others) show that fibrin, although in less amount, is also contained in the alveoli of the broncho-pneumonic foci. But to this must be added the important fact that, besides the specific organisms which commonly give rise to primary pneumonia, Fränkel-Weichselbaum's diplococci have also been demonstrated in broncho-pneumonic foci. Thus we are forced to the conclusion that, in *both* forms of pneumonia, the same infectious elements play a rôle, that there is scarcely an *essential* difference, but the preceding or non-implication of the bronchi has such an important effect upon the course and form of the disease that, where a diffuse bronchial catarrh already exists, the additional effect of the pneumococci impresses the stamp of inflammation, producing broncho-pneumonia. This view is favored by my observation of a few cases in which a fibrinous pneumonia of an entire lobe was found upon one side, and a simultaneous bronchitis with lobular foci upon the other. Whether we regard the pneumococci as having a specific action, or that they only play a rôle of simple generators of inflammation, like many other bacteria, I believe to be immaterial. No matter how these conditions may be regarded in the future, it is certain that in most

cases, at least, the *clinical* differences are sufficient to permit of a positive differentiation of the two forms.

## CLINICAL PICTURE

**Primary pneumonia** attacks children and adults alike, in the midst of complete health, as a true infectious disease with sudden high fever, frequently associated with vomiting, but often without initial chill, particularly in infants; the temperature rises rapidly to 40° C. (104° F.) and above (in one case I noted a temperature of 41.2° C. (106.2° F.) on the *first* evening), and in combination with the general signs of illness—somnolence, restlessness, mild delirium—it denotes a serious affection. However, in the first days the respiratory symptoms and even cough may be *absent*, and the physician is often puzzled over the diagnosis. Many other diseases, particularly *scarlatina*, show a similar onset, and the slight cutaneous erythema which, as a rule, is only partial, and a hyperemia and swelling of the gums and pharynx, often lead us to suspect this infectious disease. This uncertainty continues, unfortunately for the physician and parents, so long as the examination of the thorax reveals nothing abnormal, but usually not longer than from thirty-six to forty-eight hours; occasionally, however, a correct diagnosis cannot be made for several days, and in one of my cases the disease could not be determined even up to the crisis on the sixth day. Under such circumstances, as has been my experience, we think of enteric fever, meningitis, even acute osteomyelitis. Many children complain of marked hyperesthesia of the skin which renders the slightest pressure painful and which is liable to cause diagnostic errors. The nature of the *respiration* should be particularly observed. Although cough and dyspnea may be absent, the respiration is conspicuously short and much increased in comparison to the pulse frequency, and the expiration is often accompanied by brief moaning. If there is also a *diminution* of the vesicular respiration in some portion of the lung, and, in addition, even a scant crepitation which at most can only be heard upon *deep* inspiration, we may be pretty positive of our diagnosis. A differentiation may be made more difficult by the appearance, simultaneously with the onset of the disease, of serious *cerebral* symptoms, such as deep somnolence, marked delirium, temporary unconsciousness, and even severe, repeated *convulsions*, examples of which I have detailed in another article.<sup>1</sup> As to the reason for these disquieting symptoms, which in fact greatly resemble meningitis (*pneumonie cérébrale* of the French), opinions are divided. High temperature alone can scarcely be regarded as the cause; but even the effect of pneumococci and their toxins upon the brain cannot be considered, because the severe cerebral symptoms are only *introductory* phenomena which disappear with the manifestation of distinct pulmonary symptoms (dulness upon percussion and bronchial respiration) and have no influence upon the further favorable course of the disease. Such cases are

<sup>1</sup> *Vorlesungen über Kinderkrankheiten*, 1899, 10 Aufl., p. 382.



by no means rare in children and many a fortunate case of "cured meningitis" may have been a mistaken pneumonia. Here I naturally disregard those cases wherein there is a *complication* of cerebrospinal meningitis with pneumonia—a condition which will not be discussed here.

**Broncho-pneumonia.**—In *broncho-pneumonia* the initial symptoms show a great contrast to this violent onset. Usually a more or less prolonged catarrhal stage precedes the actual affection, which, if neglected, will only too frequently have serious results, particularly among the poor. In numerous cases, however, especially in small, badly ventilated and crowded rooms, and in hospitals, the propagation of the catarrh to the smaller bronchi and to the alveoli, which cannot be prevented, is only to be explained by the inhalation of bacteria, and it is here we find the greatest number of children who are affected with, and perish from, broncho-pneumonia. According to my experience the autopsy of most young children who succumb from various affections in hospitals, foundling asylums, etc., demonstrates a more or less extensive broncho-pneumonic foci in the lungs. The infectious nature of this complication may be concluded from its enormous frequency. Recently, and particularly in France, it has been maintained that the infectious organisms (streptococci and pneumococci) not only reach the lungs by means of the inspired air, but also that coli and other bacteria find their way from the intestinal canal through the lymph- and blood-channels into the organs of respiration and may produce broncho-pneumonia. This view, which is based upon the frequent simultaneous existence of enteric and broncho-pneumonic processes, has not yet been proven, and, therefore, is problematic, because the presence in the lungs of the aforesaid intestinal bacteria may be due to agonal and postmortem migration and increase, as has been demonstrated.

With the gradual distribution of the inflammation to the finer bronchi and beyond, and in addition to the catarrhal symptoms already present, there is a constant slow or rapid increase of the pneumonia, not with high and continued fever, as in the primary form, but with pyrexia which shows marked *remission* in the morning hours and in the evening may range from 39.5° to 40° C. (103.1° to 104° F.); dyspnea increases, and the cough becomes more frequent and more tormenting. These symptoms are due to the bronchitis, which distributes itself to the deeper parts, giving rise to marked irritating cough, and are much more dependent on the decreased function of the larger respiratory area than is the case in the limited primary form. It must always be remembered that in broncho-pneumonia we are dealing with a bilateral affection which consists of more or less numerous lobular foci with a constant tendency to approximation, and in which even the intervening parenchyma has become hyperemic and shows serous infiltration. But to this is added another important element: the muco-purulent exudation in the small bronchi, which impedes the entrance of air to the affected pulmonary areas, and, together with the constantly decreasing inspiratory power, finally occludes them. This explains the formation of numerous atelectatic foci in broncho-pneumonic lungs. In addition, the frequent respirations (I have

counted 80 in the minute) and the labor of the auxiliary respiratory muscles (ala nasi, shoulders, etc.) are much more intense and are accompanied by a constant moaning sound which has been aptly termed by a French author "*l'enfant crache son expiration*." I attach great importance to this symptom because it has indicated the correct diagnosis often prior to the examination. The changed relation of the respiration to the pulse frequency is also important. Normally these are in the proportion of about 1 to  $3\frac{1}{2}$ , while in the present condition there are 50 or 60 respirations to 120 or 140 beats of the pulse. That with such an important and extensive limitation of the respiratory surface stasis occurs in the venous system, as well as cyanosis and even slight edema, is readily comprehensible.

Nevertheless, we must not expect to find in all cases of this kind at once, upon physical examination, the distinct signs of consolidation of the parenchyma. So long as the lobular foci are insular, separated from one another by tissue which contains air, the percussion note shows very little change, and auscultation reveals only widely distributed, fine, moist râles and here and there crepitation, especially upon both posterior surfaces from the spine of the scapula to the base of the lungs, but also laterally and anteriorly. Occasionally I have been able to note at the onset fine crepitant râles from the left upper lobe to the lingula overlying the pericardium. Therefore, we are only capable of demonstrating physically a catarrhal bronchitis. But experience has shown that, at least until the fourth year of life, this condition *never* occurs without broncho-pneumonic foci, hence the diagnosis becomes positive. Confirmation by *physical signs* is not possible until the foci become so numerous and confluent that there is no longer an air-containing intermediary parenchyma. Only then do the physical signs of consolidation, *fine* crepitant râles in great distribution, bronchial respiration, and bronchophony become noticeable; finally, also, the dulness of the note upon percussion. This method of examination should be especially *light* over those regions which have already been mentioned, as we thus avoid the note of layers which may still contain air. All of these physical signs are *bilateral*, although not always of equal intensity, and from this fact alone, quite apart from the rapid, tumultuous onset of primary pneumonia, they differ from the unilateral symptoms of consolidation which are almost always present in the latter disease.

### COURSE OF THE DISEASE

The further *course* differs greatly. The primary form advances with continued high fever, the local phenomena remaining about the same to the crisis, which occurs usually at the end of a week, although not rarely between the third and fifth days. The fever drops temporarily, for example, from  $40^{\circ}$  to  $38.8^{\circ}$  C. ( $104^{\circ}$  to  $101.8^{\circ}$  F.), again to reach its acme in twelve to twenty-four hours. I shall recur to the deviations from this rule. On the other hand, broncho-pneumonia shows a *protracted* course, with a constant, but irregular variation of improvement and retrogression. The reason for this fluctuation

is that while a portion of the consolidated pulmonic lobule is undergoing involution, the inflammation is extending from the branch of the bronchial tree to other alveolar groups, which have thus far been intact, and new lobular foci are formed which cause the declining fever to flare up anew. Therefore the "regular course with critical defervescence" cannot occur here; the duration is indefinite, often extended from weeks to months, exhausting the strength of the child and rendering the prognosis grave. For this reason alone broncho-pneumonia is much more serious, especially in infancy, than the primary form, which ordinarily shows a favorable course if no serious complications arise. Among 153 clinical cases of fibrinous pneumonia I have lost only 8, and of these one showed at the autopsy hepatization of the entire right lung, another bilateral pneumonia and pericarditis, a third peritonitis, and a fourth multiple tuberculosis. If these figures are compared with the great mortality of broncho-pneumonia, which is an actual scourge among infants, the difference is apparent at once. Probably all physicians will coincide in this, especially if they have had an opportunity to observe cases in children's hospitals or to practise among the poor.

Although the foregoing conditions of both forms of pneumonia apply in the great majority of cases, they are by no means applicable to all. Here as everywhere in the practice of medicine there is "no rule without an exception," and it is, therefore, my object next to present a somewhat minute description of the variations from the norm.

There may be a modification of the development of *primary* pneumonia in that it may be associated with an already existing acute or chronic bronchial catarrh. In cases of this group catarrhal râles may be heard in the bronchi throughout the entire course of the disease, in addition to the physical signs of consolidation. *Critical defervescence* does not always occur in cases having a favorable termination. Among 160 of my cases crisis occurred 100 times; in 17 cases there was *lysis*, while of the remaining 43 cases no positive report can be made. In 68 cases the crisis appeared between the sixth and eighth days, less frequently it occurred on the fifth or sixth day and even between the ninth and eleventh days. There may be cases which present all of the symptoms of *broncho-pneumonia* and terminate in rapid recovery in a week, but they are doubtful, because, as I have stated, primary pneumonia may exist simultaneously with bronchial catarrh and thus render the diagnosis uncertain. On the other hand, the primary form may run an unusually protracted course, as in one of my cases wherein crisis did not occur until the seventeenth day. This prolonged defervescence was occasioned by two relapses with an afebrile interval of twenty-four hours, the lesion being in the left lower lobe.

The prolonged course in these abnormal cases is usually explained by the fact that a pneumonia resembling erysipelas in its development, creeps from its original focus to neighboring areas, for example, from the right lower lobe to the upper lobe, and thus the physical signs of consolidation at the lower portion of the right posterior surface, therefore in the area first attacked,



disappear, while in the right axillary space, and finally at the upper lobe anteriorly, there is dulness and bronchial respiration. This form, which has been designated *pneumonia migrans*, may continue for several weeks, similarly as in broncho-pneumonia, the fever declining and rising until crisis finally appears. My own cases of this group terminated favorably with a typical crisis between the twelfth and fifteenth days, but the outcome is not always so fortunate. This variety does not involve the bronchi; perhaps the condition is similar to that of erysipelas, wherein relapse is caused by a progression of the bacteria.

It is probable that to this category of *wandering pneumonia* belong numerous cases which present the same characteristics, but in a less marked form than those just described, since otherwise their deviation from the typical course cannot be satisfactorily explained. As a good example of this variety I will relate a case which I saw about the beginning of March, 1901. A robust girl, aged 9 years, was attacked at night, while returning from a children's party, with sudden, repeated vomiting, and marked chill followed by high temperature, nearly 40° C. (104° F.). The fever remained at its acme about twenty-four hours and declined in a critical manner below normal. Apart from the almost general hyperesthesia of the skin and a systolic murmur of the heart, the examination revealed nothing abnormal, therefore the attending physician made a diagnosis of influenza with an endocardial complication. The next day there was renewed fever, which rose rapidly step-wise, and upon the middle of the third day (from the appearance of the chill) it was again 40° C. (104° F.). That evening I saw the child for the first time and diagnosed pneumonia of the left lower lobe, the symptoms of which had first become manifest to the physician that morning. The systolic murmur could no longer be detected. The fever remained high almost continuously, while from the fourth to the fifth day dulness and bronchial respiration progressed toward the apex of the lung, proceeding from the origin of attack at the base. At the lower portion, however, the physical signs had decreased. In the night of the fifth to the sixth day there was crisis with copious sweating, and the affection terminated favorably.

I cannot agree with those physicians who declare such cases to be "*influenza pneumonia*" in the absence of positive proof. Quite apart from the fact that the pulmonary inflammations which depend upon pneumonia are almost always cases of broncho-pneumonia, it is very unlikely that an influenza which begins with such an intense fibrinous pneumonia can terminate in complete crisis after a course of only five days. On the contrary, I believe that these are cases of *pneumonia migrans*, with a critical defervescence of the first attack at the end of twenty-four hours, and upon the following day an upward migration with renewed fever which terminates between the fifth and sixth days in crisis, therefore unusually early. The systolic murmur (which I did not hear) was probably only an accompaniment of the high fever. This case reminds us of the type often designated "*intermittent pneumonia*," which has nothing in common with malaria, and belongs to the category of "wandering

pneumonia." These pneumonias also, notwithstanding the high fever which continues for four or five days, may have entirely latent *physical* signs, owing to the fact that hepatization proceeds gradually from the interior of the lung and becomes recognizable only when it reaches the periphery. In two instances I only succeeded in demonstrating consolidation by physical signs immediately before the crisis, when the fever began to decline. Here the absence of diagnostic symptoms continued for six or seven days and there appeared to be no other explanation for this than a progression of the hepatization from the center of the lung to the periphery (central pneumonia).

It must be further noted that occasionally on the *first* day after the crisis, even when the temperature has fallen below normal, there may be a sudden rise—39° to 40° C. (102.2° to 104° F.)—which is a source of great anxiety to the physician and parents, but the cause of which I have never fully determined. So far as I was able to observe, this sudden pyrexia was always *ephemeral*; often upon the following day the temperature again became normal and remained so.

Much more rare than a protracted course is a *short* febrile attack. I have seen three undoubted cases of primary pneumonia with crisis upon the third day, that is, in the night between the second and third days. In literature cases of even a shorter duration are reported, sometimes, in adults, a termination at the end of twenty-four hours. These so-called "*abortive pneumonias*" may actually be identical with the indistinct and varying pathologic pictures which the French have described under the name "*congestion pulmonaire aigue*," by which they do not imply actual hepatization but only temporary congestion due to extensive hyperemia and serous infiltration of the parenchyma, which, however, rapidly disappears. The anatomical proof for such a conception is not given to us by the French physicians and if, instead of this, they refer to the rapid recovery, I must quote the following case.

Max S., aged 11 years and 9 months, was admitted to the hospital upon the 27th of June with an afebrile bronchial catarrh. Upon the 30th of June sudden high fever developed—40.5° C. (104.9° F.)—with respirations of 40 per minute, a pulse of 138, cough and left-sided pain on respiration; below the left scapula respiration was indistinct. On the 1st of July there was dulness and bronchial respiration with crepitant râles, temperature 40° to 40.4° C. (104° to 104.7° F.), respirations 40, great debility at night, sputum decidedly *rusty*. The following morning (beginning of the third day), after profuse *sweating* and good sleep, there was a condition of comfort. Temperature 37° C. (98.6° F.), respirations 25, pulse 80. *On the 4th of July the dulness had disappeared; there was only harsh breathing with isolated crepitant râles.*

In this case, which is at the same time a good example of the onset of crisis during the night between the second and third days, no expert would doubt that he was dealing with an actual pneumonia, particularly as rusty sputum was often present,—a rare occurrence in *infancy*. Nevertheless, the physical signs of consolidation had disappeared on the fifth day after the onset of the disease. Such a course is very unusual, for, as a rule, at least eight to

ten days elapse before evidences of consolidation have *completely* disappeared. If the condition continues for a longer period we must consider that we may be dealing with a *pleuritic* condition, since a complication with pleurisy occurs in children as well as in adults. I must also mention that in three children I have observed the disappearance of physical signs even *before the advent of the crisis*. Although there was continued high fever—40.6° C. (105.1° F.)—and other pathologic phenomena were still noted, the dulness had disappeared and fine crepitation was superseded by mucous râles two or three days prior to complete crisis.

Analogous observations in adults have been reported by Grisolle and Sidlo. Whether in these cases a bacterio-toxic influence maintained the fever for some days, or whether anatomical changes persisted which were not demonstrable by physical examination, cannot be determined.

### PROGNOSIS

That primary pneumonia offers a much more favorable *prognosis* than broncho-pneumonia, and the reasons therefor, have already been discussed. Nevertheless we must never be too positive in our conclusions. We may expect a favorable outcome in children previously healthy and with a limited extension of the disease, and these cases fortunately preponderate. Among my 160 cases 153 were affected only in *one* lobe of the lungs and most frequently the lower lobe (the right upper lobe 33 times, the left upper lobe 4 times). Implication of the entire lung (which occurred in only 3 instances) and consolidation of *both* lower lobes, is rare, and when such a condition occurs the prognosis is naturally more unfavorable. This is also true when pneumonia appears in a child already ill, and particularly when it develops in the course of an infectious disease. While the pneumonia which occurs as a complication in tuberculosis, measles, influenza, scarlatina, whooping-cough and enteric fever is usually the broncho-pneumonic type, other forms may be noted. In most of these infectious diseases I have observed cases of fibrinous pneumonia, and this condition has often been demonstrated at autopsy. In a child of 12 years, suffering from *enteric fever*, with an almost continuous temperature of 40° C. (104° F.) or over, which was not influenced by treatment, the left lung was found to be completely consolidated, and in the midst of this thickening, upon the lower boundary of the upper lobe, there were two insular foci the size of a bean and of a hazelnut. Another case, a boy, aged 4 years, who was attacked by pneumonia during convalescence from a *diphtheritic nephritis*, succumbed nine hours after a fulminant onset of the fever, and the autopsy showed hepatization of the entire right lower lobe, with a pale, flaccid heart, which, unfortunately, was not examined under the microscope. Such an examination would unquestionably have demonstrated the cause of the rapid and fatal course of this very limited pneumonia.

As has already been stated, *pleurisy* is very often a complication of fibrinous pneumonia in children, just as in adults. In older children this com-



plication is manifested by pain upon breathing or coughing, or when lying upon the affected side, and by physical signs upon palpation and percussion of the intercostal spaces. But this complication is of greater importance in infancy, since a *purulent* exudate is much more likely to be present than in later years, and this may develop with such rapidity that in five to seven days there is a well-marked empyema. In *infants* the pericardium may be affected and a purulent *pericarditis* has frequently been present of which there were no definite signs during life.

In the foregoing case of typhoid pneumonia the autopsy revealed two sequestered foci in the midst of the consolidated parenchyma. They were readily distinguished from the surrounding brownish red hepatization by their yellowish tint, demarcated by a dark red line. If the case had continued longer there would undoubtedly have been either a *gangrenous necrosis* or an *abscess* in these foci,—two methods of termination which, at least in my experience, are exceedingly rare in children otherwise healthy. Larger abscesses, which may be recognized during life, I have noted but twice: in a girl of 7 and in another of 11 years. In both patients cure followed the spontaneous expectoration of profuse amounts of pus. It is of interest to note that in one of these cases a pneumonia appeared abruptly after the fifth injection of 0.001 of tuberculin. The child was not tuberculous, but injections were instituted as a test by the wish of the exceedingly anxious parents. However, a very instructive proof was furnished of the inflammatory effect of tuberculin, which in this unfortunate child caused a prolonged illness. I am unable to state whether fibrinous pneumonia of children may terminate in a *chronic* condition, apart from the development of a pulmonary abscess, especially that, by the formation of new interstitial connective tissue, there is an induration of the affected lobe which finally causes bronchiectasis by contraction of the newly formed tissue. The cases which have been designated “interstitial pneumonia” (Steffen and others) are not sufficiently clear in their development to be definitely regarded as a termination of a “croupous pneumonia”; at least I have not been able to demonstrate this origin in a large number of cases of pulmonary contraction in children. The development of this condition from a chronic *broncho-pneumonia* however cannot be doubted. Nor will I deny that a pneumonic consolidation under some conditions, not always apparent, may require an unusually long time—several weeks, or even months—for complete resolution. A case of this kind wherein two months elapsed before all of the physical signs of consolidation had disappeared, has been recorded by me;<sup>1</sup> but even here I could not be positive whether small pus foci, which could not be demonstrated by physical diagnosis, retarded recovery. However, certain symptoms (remittent fever, emaciation, occasional blood-streaked sputa which later became purulent, but in which threads of tissue could never be found) seem to justify this suspicion. Although I have never observed a case of pure fibrinous consolidation which ran such a chronic

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<sup>1</sup> “Vorlesungen über Kinderkrankheiten,” 10 Aufl., 1899, p. 392.

course in an otherwise *healthy* child, nevertheless, it must be noted that children who have once suffered from pneumonia are predisposed to further attacks and that even a *relapse* may follow quickly. A child, aged 4 years, suffering from pneumonia of the left lower lobe, was again attacked in the same lobe immediately after the defervescence. Again, in a boy of 3 years, I saw a pneumonia of the right succeeded by a pneumonia of the left lower lobe in the course of fourteen days, with a favorable termination.

### COURSE

In contrast to the almost continuously tumultuous course of the fibrinous form, **broncho-pneumonia** shows from the onset a decided tendency to protraction, wavering between improvement and aggravation. There is frequently a transition to a *chronic* condition which may last for months and lead to the development of other serious affections. It cannot be denied that often complete recovery may ensue after the disease has continued for months; I have repeatedly demonstrated this fact clinically and also by anatomical investigation in children who succumbed to other diseases soon after their recovery from broncho-pneumonia. The possibility of complete resorption of the fatty contents of the alveoli is therefore evident. But in the course of this disease there are so many threatened dangers which originate from the reaction of the prolonged limitation of respiration upon the entire infantile organism that the *prognosis* should always be made with the greatest caution. As sequels of this condition there may be insufficient oxidation of the blood and stasis of the venous system, anemia and general debility, or more or less extensive edema, and even dilatation of the right side of the heart. The last change I found particularly developed, coincidently with partial fatty degeneration of the myocardium, in a few cases of broncho-pneumonia which occurred as a complication of whooping-cough, probably because the frequent paroxysms of cough increased the resistance which the heart had to overcome from the long-continued consolidation of the pulmonary tissue. Under such circumstances death may occur unexpectedly as a result of syncope. It must also be remembered that in such a prolonged febrile disease the appetite is much decreased and in infants the ingestion of food is influenced by the dyspnea, which makes nursing difficult. When such a child attempts to feed from the breast or a bottle it will be noted that he lets go of the bottle after a few moments to breathe. It may therefore become necessary to feed the child with a spoon or from a cup. Even then the food may be rejected, and as a consequence the debilitation already present will increase. Here the weakness of the inspiratory muscles plays the most important rôle. The respirations become more rapid (I have counted over 100 in a minute), and more superficial. They may at intervals resemble the Cheyne-Stokes type, and it is clear that these deficient inspirations may seriously influence the existing atelectasis. The power to cough is lessened and this symptom finally disappears—an exceedingly unfavorable sign; for although children from 8 to 10 years of age rarely eject sputum, they raise it to

the level of the pharynx and swallow it. When cough disappears, this means of expulsion is no longer possible, and the widely distributed large and small moist râles, and in part the crepitant râles, denote an over-filling of the bronchial twigs with mucus. Carbonic acid poisoning finally develops as a consequence of this respiratory insufficiency, and these little unfortunates become somnolent and perish, occasionally during convulsions. In every *extensive* broncho-pneumonia the physician must consider these dangers, and he will be fortunate to have even a portion of the cases terminate in complete recovery, with a gradual cessation of the threatening symptoms.

The *environment* of the patient has a great bearing on the prognosis. Those who are most endangered, as I have already indicated, are the inmates of foundling institutions, hospitals, or those who live in badly-ventilated, crowded rooms. Usually there is another deleterious factor, the *continuous recumbent posture*, which is a further hindrance to respiration, giving rise to hypostasis of the lower lobes. Among the well-to-do improper hygienic conditions are not common, so that the affection does not develop to such a serious extent, and recovery occurs more readily. Under favorable hygienic conditions cases which I considered almost hopeless have terminated in recovery notwithstanding many fluctuations. The *previous health* of the child has an important bearing on the course of broncho-pneumonia. Children in whom the affection develops from a simple bronchial catarrh are much less endangered if they were previously healthy than weak, anemic children, especially *rachitics* with a prominent chicken breast. In the latter we are not dealing alone with a lessened resistance, but, by the limitation of the cavity of the thorax a mechanical factor is added which decidedly increases the danger. Also, in these cases, extensive atelectases are particularly frequent, and for this reason every bronchitis, and certainly every broncho-pneumonia, is to be regarded from the onset as more serious than in a previously healthy, non-rachitic child with a normal chest. The same is true, although not always to the same extent, of broncho-pneumonia which occurs in combination with *debilitating* diseases, especially a prolonged *intestinal catarrh*,—a condition which I have already mentioned as being frequently referred to the entrance of intestinal bacteria into the respiratory tract. That *tuberculous* children are greatly endangered by broncho-pneumonia is self-evident, and the condition is all the more serious in these children if there is a predisposition to bronchial catarrh.

Among the acute diseases with which broncho-pneumonia may be associated—very frequently in a dangerous form in consequence of the accompanying catarrhal affection—*measles* must be especially mentioned. In rare instances I have seen this complication in the prodromal stage, or simultaneously with the development of the exanthem, but, notwithstanding the very intense symptoms, the affection is less serious at this period than later, after the eruption begins to fade and the fever falls by crisis. If fever again develops in the succeeding days we should at once think of broncho-pneumonia, and a careful examination of the chest should always be made. This rise in the



temperature may be due to some other condition, as an otitis media, but a positive knowledge of the condition of the lungs is imperative. Therefore it is absolutely necessary to take the morning and evening temperature even for days after the defervescence, so that we may recognize the first symptoms of fever and act accordingly. *Thermometry* is of inestimable value in this condition.

Next to measles, *influenza* and *whooping-cough* must be designated as the most frequent causes of broncho-pneumonia. By far the majority of children who succumb to these diseases suffer from inflammatory lung affections which are not common in the other infections. But even in *scarlet fever* and *enteric fever* we cannot be too careful, and in enteric fever in particular the lungs must be thoroughly examined, as a general debility may affect the inspiratory muscles to such an extent that the râles heard in the smaller bronchi only become distinct upon *deep* inspiration, which can usually only be produced in older and more intelligent children.

In *diphtheria* also we must reckon with this complication, whether it runs its course with or without implication of the *upper* respiratory passages. Therefore, in diphtheritic croup a serious symptom is a sudden rise of the noisy respiration from the normal frequency to 40 or 60 per minute. We may then be certain that broncho-pneumonia has developed; that it is not alone due to a propagation of the inflammation from the larynx and trachea, but also to the aspiration of diphtheritic products from the upper respiratory passages, as was proven by a case in which I found extensive broncho-pneumonia, with partially septic, decomposed foci, and complete integrity of the larynx and trachea. It is, therefore, not justifiable to ascribe the cause of this fatal complication to a tracheotomy, as has frequently been done. Tracheotomy alone has very little to do with the condition; it may only prove serious in consequence of "deglutition"—an accident which is not infrequent—whereby a portion of the ingesta finds its way to the respiratory passages and is aspirated into the bronchi; therefore it plays the same rôle as in other pathologic conditions which favor such a process, particularly of the cerebrum. It is, then, the irritation caused by the particles of food which find their way into the bronchi which produces the serious pulmonary condition known as "*deglutition pneumonia*."

I have always been under the impression that the broncho-pneumonias which occur as a complication of the foregoing infections are much more serious than those forms which develop from a simple bronchial catarrh, not only on account of the dangers which arise and which are probably increased by the infectious nature of the condition, but also from their great tendency to an exceedingly protracted course which may continue for months, and—what I must emphasize particularly—because of the pathologic picture which they present which closely resembles *tuberculosis*. Tenacious cough, dyspnea, sighing expiration, remittent fever with evening exacerbation, and in particular the increasing debility and emaciation, which in some of my cases was so marked as to be almost skin and bones, readily justify the suspicion

of a caseous decomposition of the broncho-pneumonic infiltrates. In this condition the physical signs, the catarrhal and crepitant râles, continue undiminished, while dulness of the percussion note in the areas originally attacked may disappear, to become prominent in other portions which were previously exempt. This variation, the conditions of which I have already described, is even a favorable sign in so far as it shows the resolution of some portion of the infiltrate. But there are frequent cases in which this variation does *not* occur; on the contrary, dulness, feeble or indistinct respiratory murmur, bronchial respiration and crepitant râles are constantly present in the same area. Under these circumstances the diagnosis of "phthisis" forces itself upon us, particularly when the seat of this chronic consolidation is not only at the lower portion of the lungs, but also in one of the *upper lobes*. That the diagnosis of phthisis may be justified is undoubted; every physician knows that tubercle bacilli find a particularly favorable soil in broncho-pneumonic areas of consolidation, and a certain percentage of these chronic cases succumb from tuberculous caseous decomposition of an infiltrate. The diagnosis is all the more uncertain because of the difficulty in obtaining sputum for examination for bacilli. Only recently I experienced this trouble in a child who was approaching the age of puberty; in spite of all efforts some months elapsed before we succeeded in obtaining sufficient expectoration to demonstrate the presence of the pathogenic microorganism. The difficulty is even greater in *young* children, where we may be able to attain our object only by cautious removal of the contents of the pharynx after an attack of cough. The search in the feces for bacilli which have been swallowed appears to me to be a problematic process. Dependent upon such an investigation the diagnosis may remain uncertain for some time, and many a case may be regarded as hopeless. Fortunately, complete recovery may nevertheless occur, and we must, therefore, assume that the threatening symptoms probably do not depend upon a tuberculous degeneration but only upon the unusually long existence of the insufficient respiration, and that their effect upon the general condition is dependent upon the broncho-pneumonia. In the hospital I have seen such children years afterward so robust that I would never have recognized them had not their parents brought the previous admission card, on which the diagnosis had been noted as an aid to my memory. In private practice we may be even more hopeful. I recall the child of a landowner in the neighborhood of Berlin, referred to me with a diagnosis of "*intermittent fever*," who at once impressed me as a person suffering from pulmonary disease on account of the emaciation, dyspnea, cough, and markedly remittent fever. After recovery from broncho-pneumonia some months previously the child retained an extensive consolidation of the left upper lobe. The patient recovered after careful nursing and a residence for two winters in the south. In cases of this kind are we to assume a "cured tuberculosis" after bacilli have been manifest? And particularly after such an advanced stage as might be assumed from the physical signs? I believe this to be absolutely unjustifiable. On the other hand

it is quite possible that in this consolidation small pulmonary abscesses occur, in that the alveoli which are filled with epithelium and new cells rupture here and there and form larger cavities by confluence, which, however, escape detection on account of their slight extent. As a matter of fact, in chronic broncho-pneumonia I have observed abscesses the size of a hazelnut; for example, a child, who remained in the hospital nine days, twice had a temperature of  $38^{\circ}$  to  $39^{\circ}$  C. ( $100.4^{\circ}$  to  $102.2^{\circ}$  F.) and presented a normal or subnormal temperature in the intervals. An abscess formation is particularly favored in those *broncho-pneumonias* arising from small foreign bodies which find their way into the bronchioles. I have seen two such cases, in which, after the symptoms of a broncho-pneumonia had persisted for months and a fatal termination seemed unavoidable, a glass bead and a bean respectively were expectorated with thick pus or pus containing blood. The first child recovered; the second escaped my further observation. The preceding case, however, shows that even without such a cause, the formation of small abscesses is possible, and although I do not maintain that small pus foci are present in all cases which resemble phthisis and terminate in recovery, nevertheless it must be admitted that in at least a portion of the cases the serious prolongation of the illness may thus be explained, especially when *hemorrhagic sputum* is sometimes present in addition to the above mentioned symptoms. I have observed this condition a few times in older children, a fact which would still further justify the suspicion of phthisis. Whatever our opinion, we must never omit a careful microscopic examination for pulmonary tissue if sputum is at all obtainable.

### PROPHYLAXIS AND TREATMENT

The *treatment* of infantile pneumonia, which we will now consider, is a subject concerning which there is much diversity of opinion. Although, as in therapeutics generally, there may be unanimity as to certain fundamental principles, nevertheless, individual views, insufficient criticism of our own and other experiences, blind confidence in new remedies and their application, play so great a rôle that it would be a difficult and thankless task for me to describe the therapy which is largely dependent upon the experience of others and is not subject to my *own* control. I prefer, therefore, to discuss only such therapeutic measures as I have found of value in my long hospital and private practice, whereof my observations, fortunately, coincide in the main with those of the most experienced investigators.

In primary fibrinous pneumonia the *prophylaxis* is as little efficient as in most of the infectious diseases; nor are preventive measures at our command in the epidemics and endemics of pneumonia, which, however, are relatively rare in children and fortunately are limited in extent. Inversely, in the treatment of broncho-pneumonia, which must be directed to the preceding catarrhal condition, prophylactic measures are of avail. This is especially true of bronchial catarrh in *infancy* because then there is a tendency



for the catarrh to propagate itself with surprising rapidity to the medium-sized bronchi and further downward. Here great care is necessary. The belief of many women that, even during storm and rain, fresh air is best for their children, is difficult to dispel, and many broncho-pneumonias, particularly among the poor, are due to exposure and insufficient clothing. I cannot agree with those physicians who advise that children suffering from whooping-cough should be kept in the open air even in bad weather and in winter, because not only does this method not benefit the child, but the catarrh which accompanies the condition may rapidly lead to grave broncho-pneumonia. It is important, particularly in the first three years of life, to protect children from "taking cold" (*sit venia verbo*), and they should be put to bed as soon as there is even slight fever. By an observance of these rules much danger may be averted.

**Treatment of Primary Pneumonia.**—We will now turn to the treatment of *primary fibrinous pneumonia*. It has been already stated that in the first days of this disease a diagnosis is often impossible and there is then only *one* requirement to fulfil, which is indicated by the high *fever*. It is a matter of course that the sick child must be kept in bed; the thirst must be relieved by cooling drinks (lemonade, orangeade, etc.), while the decreased appetite or refusal of food necessitates but small quantities of milk or starchy substances (gruel or grits). Applications of ice to the head are advisable, particularly if there is severe headache with somnolence and delirium. Local withdrawal of blood from the head should only be considered when there are pseudo-meningitic symptoms, which have previously been detailed. But even in these cases I must enjoin caution, because blood-letting is *unnecessary*. As soon as the pneumonic symptoms become prominent (after two or three days) the cerebral symptoms grow indistinct or disappear spontaneously; therefore a little patience should be exercised. But I must admit frankly, from my own experience, that with the appearance of fulminant symptoms (convulsions, sopor) there is a great temptation for energetic action, and the physician, lest he be neglectful, will apply leeches to the head to avoid a possible meningitis. It is true he will not do much injury to a normal child, provided after-bleeding is not profuse, but it must be observed that in children an arrest of the bleeding is not always easy and, as a consequence, there may be a very undesirable diminution of strength. In any event, it is better to limit the treatment to the application of ice and patiently to await the further course of the disease.

Even after the diagnosis has become manifest we can do little more than wait. In the treatment of pneumonia there is one fundamental law, namely, not to do too much. In children, much more than in adults, the principal danger is the appearance of *cardiac weakness*. Therefore, everything which favors this calamity must be avoided, and for this same reason I am seriously opposed to the repeated employment of internal antipyretics, such as antipyrin, phenacetin, and aspirin, because in small doses they are ineffectual, and large and repeated doses may produce serious results. Quinin (0.3 to

1.0 [5 to 15 grains] given during the afternoon) is much less depressing, but in my experience it lowers the temperature for only a few hours, and I have finally decided to abandon internal antipyretics and to limit myself to *hydropathic compresses* of the thorax. I am also opposed to the *cool* or *cold* baths recommended by some authorities, first, because I regard all “energetic” remedies as unnecessary, and, secondly, on account of their depressing influence upon the cardiac power of the child which cannot be estimated beforehand—a condition which I have determined in enteric fever of infancy. In their stead I apply wet packs to the upper part of the body from the axilla to the navel, wrung from water of about the temperature of the room—never from cold water—and changed every hour. I have no objection to *lukewarm* baths, although they exert but a transitory influence on the temperature. In very excitable children, however, they often have a quieting effect which cannot be overestimated. In the winter the sick-chamber should not be overheated, the temperature never above 15° C. (70° F.), while in summer a room well ventilated, and cooled by means of ice-filled vessels is preferable. Instead of packs, hourly spongings of the entire body with cold water have been suggested and are of service, although I prefer the packs.<sup>1</sup>

*Internal* treatment generally may be dispensed with, since there is no remedy at our command which has an influence upon the pneumococci nor upon the infiltrate which fills the alveoli; nor can we prevent the spread of the inflammation (*pneumonia migrans*). I have limited myself to the employment of hydrochloric acid (0.5–1.0 to 120.0), because the taste of this mixture is unobjectionable, it is readily borne by the stomach, and also for the reason that in private practice, even in our “highly civilized” time, we can scarcely avoid a bottle of medicine.

In my experience the majority of cases run a favorable course under this simple treatment. Nevertheless, some of the symptoms may require more energetic measures. The cough, which in adults frequently requires narcotics, is rarely so distressing in children as to indicate such remedies. However, should the cough be uncontrollable or, as before mentioned, the little patient be very excitable on account of hyperesthesia, I do not hesitate to administer small doses of morphin (0.001 to 0.002) once or twice daily, from which I have seen good results. When older children complain of *active pleuritic* pains on respiration and cough, it is advisable to use from 6 to 10 *dry* cups (according to the age of the patient) over the painful area. These usually have an excellent effect, and may be repeated as often as their use is indicated, since they remove no blood from the body. I have seldom had recourse to these measures, nor have I employed *general* blood-letting by venesection, but this remedy, which was formerly so commonly employed in pneumonia of adults, has recently been adopted in children and even infants in cases which present the symptoms of marked over-filling of the

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<sup>1</sup> I advise a temperature of about 60°, instead of 70°. A window should always be open, but direct draught avoided. The good influence of fresh air is undoubted, and a much colder temperature is strongly advised by Northrup.—EDITOR.

heart (cyanosis, great dyspnea, small pulse), and particularly when there is also a very extensive or even bilateral pneumonia. Literature contains reports of a few such fortunate cases. Thus far I have not found it necessary to employ venesection, but from my experience in the treatment of infantile broncho-pneumonia, which is soon to be detailed, I believe that even in these rare cases the liberal employment of dry cups over the entire anterior or posterior surface of the thorax will be sufficient alone, and will not decrease the strength of the patient.

Special consideration must be given to the danger of *cardiac asthenia*, which is to be feared in pneumonia as in all infectious diseases, and here I must caution against the employment of depressing remedies. This danger may occur in any severe pneumonia, even without such drugs, not only in weak children or those who have been previously ill, but also in strong, robust children, and particularly near the time of *crisis*. The longer the period of fever the greater is this danger. With the rapid fall of temperature to the normal or subnormal there is a tendency to collapse, with a small, slow, or, rarely, a very rapid pulse, great weakness, and a sensation of cold with or even without profuse sweating. This condition is distressing to those about the patient, but is less serious than would appear; at least I have never seen a child perish under such circumstances. On the contrary, I have always been able to restore the cardiac power by the employment of good wine (a tablespoonful every half hour), strong meat broths, or a mixture of milk and brandy. Such stimulants as the subcutaneous injection of ether or camphor were seldom indicated in my cases, but they may prove of service where there is a prolonged course and marked debility, as in a child aged 3 years, whose temperature fell to 34.8° C. (94.6° F.) at the crisis. Although such a grave collapse is rare, it is well to forewarn the parents and to relieve their anxiety.

**Treatment of Broncho-pneumonia.**—As a rule, therefore, the treatment of primary pneumonia is expectant; only with the appearance of certain conditions already detailed, which force themselves into the cyclic course of the disease, are more energetic measures justified. Quite different is the condition in broncho-pneumonia, which, by its course and prognosis, does not permit of a delay in treatment. Although here as in the primary form we do not possess remedies effectual in the resolution of the lobular consolidation, or which will prevent the formation of new foci, nevertheless, we have an important object to attain in rendering the bronchi *permeable* to the inspired air and thus prevent the development of multiple atelectases.

Emetics and expectorants have been regarded as the most suitable remedies for the fulfillment of this condition. Whether the latter are as serviceable as is expected of them is uncertain. In mild cases infusion of ipecac (0.2–0.5 to 120), or powdered ipecac (0.01) with calomel (0.02–0.03)—remedies which have always been advised in the febrile bronchial catarrhs of children—may be given every two hours. In well-developed broncho-pneumonia I strongly advise repeated small doses of *tartar emetic* in the



first four or five days of the disease, provided the strength of the child is good. My experience with this remedy has not justified the fear of many physicians, and in small children I have found this drug to be particularly valuable under certain conditions. If, however, the child is already debilitated by the disease or is simultaneously suffering from diarrhea, as is frequently the case, I believe tartar emetic to be contraindicated. For this reason the remedy should seldom be used in hospital practice and especially in very young patients, but robust children in favorable surroundings derive unquestioned benefit from such treatment, particularly when it is instituted during the first days of the disease. It is true it is not always easy to determine the exact strength of the patient, and here the "professional tact" of the physician will play a greater rôle than theoretic considerations, which are often misleading in the individual case. However, it is always in our power to discontinue the remedy with the appearance of depression, and for this reason the physician should visit the child at least twice daily. I am also particularly cautious in the employment of tartar emetic in my practice among the poor and in the hospital, because the anxious, ignorant mother frequently disregards the careful directions of the doctor and may thus cause a dangerous collapse. My method is as follows: Of a solution of tartar emetic (0.05–0.1 to 120, according to the age) I first give a teaspoonful *every hour* until it induces vomiting. The drug is then administered every two hours. Should vomiting occur after the first teaspoonful the remedy is given at intervals of two hours, and if emesis follows each dose, or there is *diarrhea*, the treatment is *discontinued*. If vomiting does not occur even after three hourly doses it is best to continue the treatment with intervals of two or three hours in order to avoid a cumulative effect, which may be exceedingly difficult to control. I have seen no serious result from this treatment, cautiously continued for days, and very often it exerts an unquestionably favorable influence upon the entire course. The vomiting which almost always occurs after the first doses and the expulsion of bronchial mucus thereby induced, can scarcely explain the success of the treatment; on the contrary, there must be some specific action upon the respiratory mucosa for which there is not a ready hypothesis.

In cases which are unsuitable for treatment with tartar emetic, wherein at least temporary relief of the over-filled bronchi and free respiration are necessary, an emetic of powdered ipecac is advised (1.0–2.0 in 30.0 of distilled water, with 15.0 oxymel scillit., a teaspoonful every ten minutes until vomiting occurs). We must proceed cautiously even with this remedy because vomiting may in itself prove exhausting to debilitated children. In fact, in the *advanced* stage of the disease we must be exceedingly careful with all emetics, and if there are symptoms of great debility, somnolence, etc., this treatment should be avoided. In such cases, and also those in which tartar emetic cannot be employed, a decoction of senega or polygala amara (5 to 100 with liquor ammon. anis 1.5 and syrup. alth. 20, a teaspoonful every two hours) are remedies which increase the cough and therefore increase

expectoration. To me the most alarming symptom is the disappearance of cough, followed by the constant noisy respiration which indicates the dangerous over-filling of the bronchi with secretion.

In addition to internal treatment *hydropathic packs* of the chest are of value (as in the treatment of the fibrinous form). I have had such favorable results from this method in broncho-pneumonia that I unhesitatingly recommend it above all other remedies. A towel of a size corresponding to the circumference of the body is dipped in water of about the temperature of the room, wrung out, placed lightly upon the thorax so that no pressure is exerted, and wrapped around the entire trunk from the axilla to the navel, but leaving the arms free. Over this a layer of cotton is placed and a final protective cover of oiled silk or rubber. This pack remains for two or three hours and is renewed continuously during the day and also through the night. Should sweating appear, water at a temperature of  $26^{\circ}$  to  $27^{\circ}$  C. ( $78.8^{\circ}$  to  $80.6^{\circ}$  F.) should be substituted for the cool water. As to the effect of this treatment there may be some dispute. In my opinion several factors must be considered: first, the stimulation of deeper inspiration by the contact of the cool pack; second, a gradual stimulation of the skin which is evinced by redness, small papules and desquamation of the epidermis; finally, the evaporation of the water which moistens the air around the child. A serviceable adjunct to this very beneficial moisture is a spray-apparatus placed upon a table near the child's head, or a tea-kettle which is kept steaming day and night, or, best of all, the vapor arising from a solution of sodium chlorid or sodium carbonate. The physician must never neglect to advise those in charge of the suffering child to *carry it* upon the arm for thirty minutes at intervals of two hours, because an unchanged position upon the back or even upon the side favors hypostasis and thus still further limits the respiratory area.

Moderate sweating does not prevent the continuance of the foregoing treatment, but I advise its immediate abandonment as soon as sweating becomes general or copious. Although it is a rare occurrence, nevertheless sudden collapse, similar to that previously mentioned, has appeared under such circumstances, which I was forced to combat with wine, dry friction of the skin, and even with stimulating drugs. In any event the relatives of the child must be prepared for this emergency, for in this condition even more than in primary pneumonia it is necessary to avoid asthenia, which is so serious on account of the decreased inspiratory power and the production of venous stasis of the brain and other organs. I have already stated that this collapse in broncho-pneumonia, which is due to a decrease of cardiac power, may also arise in consequence of the prolonged fever and the insufficient respiration, and the first signs of this dangerous condition require energetic action by the physician. All other considerations are secondary to this indication to improve the weakened energy of the heart.

Among the internal remedies which must be considered I advise, in addition to strong wine or cognac, a combination of camphor with benzoic acid

( $\bar{a}\bar{a}$  0.05–0.2) and sacch. alb. 1.0 in powders, one to be given every two hours, or when their internal administration is difficult, or a rapid effect is necessary, subcutaneous injections of camphor are serviceable. Instead of the generally recommended oil of camphor I prefer a solution of camphor in ether somewhat like the following: camphor 0.6–1.0 with sulphuric ether 10.0, of which a Pravaz syringeful may be injected according to indications. The injections may be given three or four times daily until there is distinct improvement. The red infiltrations which are occasionally visible at the point of injection never have had any serious effect on my cases even when multiple; on the contrary, in ten to twelve days after the formation of a yellowish line of demarcation they disappeared, leaving a sharply circumscribed loss of substance which healed rapidly.

These symptoms of collapse due to cardiac weakness, just described, must not be confounded with those which arise from extensive confluent foci which greatly limit the respiratory area. I admit that under these circumstances there may be a *simultaneous* implication of the energy of the heart and a complicated pathologic picture is thus produced which makes a choice of remedies exceedingly difficult. Under these apparently desperate circumstances rapid action is necessary. There is little to be lost and everything to be gained, and in fact, in a number of these cases I have succeeded by somewhat drastic measures in bringing about a change for the better and final recovery. To produce a powerful derivative effect externally, even in very small children, I have had as many *dry cups* applied to the thorax as space permitted. This method of derivation I prefer to vesicants and mustard, which were formerly so much employed, primarily for the reason that they are less effective, and further, because their improper use may produce very undesirable sequels. As this procedure does not cause loss of blood there is no essential deteriorating influence upon the strength of the child, therefore I do not hesitate to apply a number of dry cups even with an *abrupt* onset of the disease, and believe that thereby a favorable effect has been attained. However, with the usually gradual development of the affection this method should not be employed. A second measure which has often proved serviceable in these cases is a *warm bath*, lasting about five minutes, in water of a temperature about 29° C. (84.2° F.), to be followed by a rapid ablution of the head and shoulders with water about the temperature of the room. After careful drying the child is enveloped in a woolen blanket, placed in bed, and given a few teaspoonfuls of wine. The sweating which may soon occur should be stopped so soon as it becomes excessive and threatens to debilitate the patient. In the hospital this energetic treatment may readily be instituted, but I must not conceal that in private practice it frequently meets with the resistance of the parents, and because of the uncertainty of success the physician himself sometimes hesitates to face the reproach of the relatives in the event of failure.

I have attempted, in so far as my experience justifies, to present the indications for the treatment of infantile pneumonia, and the conditions



suitable for its fulfilment. But I know only too well that these therapeutic measures will not always appear sufficient when it is necessary in the individual case to decide in favor of any particular method. As a matter of fact, I believe it to be impossible to describe a method which will prevent therapeutic variation. Although the cases in general which the physician has to combat have much in common, nevertheless there are such essential differences that his judgment alone must determine which therapy will be the most efficient, and at the same time he must observe the fundamental law, that it is not the disease but the individual that is to be treated. Here error may readily occur, not alone to the tyro in practice, and, unfortunately, cannot be avoided. Only with experience can the physician hope for surety in treatment.

[Very few therapeutic remarks are herewith ventured in connection with this clinical masterpiece of the famous teacher of several generations. Vesicatories should be avoided, for they rob the patient of rest and sleep. The treatment with tartar emetic is risky. It may prove depressing, and cause diarrhea. It is the treatment of (Teschier and) Rasori, the remodeler of Brownism. Eighty and sixty years ago it was quite popular in Europe, and in my clinical work nearly 60 years ago, and during my first decade in New York fifty years ago I employed it much. Afterward I substituted for it the oxysulphuret of antimony but gave it up on account of the occasional depression caused by the antimony even in that mild form. Whatever weakens should be avoided. Every day of a pneumonia, both fibrinous and broncho-pneumonia, adds to the debility of the patient and of his heart. Mild or full stimulation should not be postponed until "the necessity turns up," that means often until it is too late. From that point of view the master recommends alcohol at an early date. My personal experience speaks against it in the first few days, but small doses of digitalis or spartein are well tolerated. The 1 drop dose of liq. ammon. anisatus is too small for any effect. Four or six drops are a dose for the smallest child. The carbonate of ammonia may upset the stomach. Benzoic acid has that effect too often, it has a very bad taste and should be avoided. Camphor is too little employed by us. Camphor water, or camphor in diluted mucilage is easily tolerated, and acts beautifully as a stimulant and expectorant. Its solution in ether as a subcutaneous injection is reprehensible on account of intense pain; the camphor oil of the Pharmacopeia is too thick. The best solution is in sweet almond oil 1:4.—EDITOR.]

# RÖTHELN, RUBELLA, GERMAN MEASLES

By CH. BÄUMLER, FREIBURG

THE conditions of hospitals in which eruptive infectious diseases are treated, as a rule, are such that the student only sees in adults those diseases which, later on in life, he encounters principally among children. This also has its advantages. On the one hand, many peculiarities are noted regarding the appearance and course of eruptive diseases which occur particularly in infancy, such as measles, scarlatina and rubella, which are then of value in judging the condition in children. On the other hand, the study of the previously mentioned diseases in adults is important in a differentio-diagnostic respect, as in them conditions arise in which the decision in the individual case is of far greater importance than in the case of children.

The following history of *rötheln* will serve as a text for the description of the disease:

H. J., locksmith, aged twenty-one, was admitted to the hospital May 18, 1900. He had previously been well, and the day prior to admission, without any other symptoms, with the exception of a mild coryza, noted red spots upon his arms and chest.

Upon admission he had an axillary temperature of 100° F., pulse 100 per minute, and upon the entire face, upon the neck, trunk, and upon the arms, as well as upon the thighs, a light red macular eruption was present. The face showed no diffuse reddening and on the rest of the body the skin was pale between the indented, here and there confluent, spots. At many points in the eruption, the somewhat swollen cutaneous follicles were reddened. The eyelids were swollen, the conjunctiva moderately injected, the upper and lower lips were covered with spots, and the pharyngeal mucous membrane was somewhat reddened; there was no decided swelling.

*Bilateral enlargement of the superficial cervical glands.*

Nothing abnormal in the organs of the chest, *no catarrhal phenomena.*

*Splenic dulness extended 6 cm., border of spleen could be felt.*

Urine free from albumin, *no diazo-reaction.*

Temperature, eight o'clock in the evening, 99.7° F.

May 19th: The eruption had almost the same appearance as the day previously. The record states:

Upon the lower legs some few individual spots are noted, the palms and dorsum of the hands are free from eruption, the cervical glands have become somewhat larger, and the inguinal glands appear more or less swollen. The temperature in the morning is 98.6° F., pulse 88, respirations 22.

Evening temperature 98.9° F.

May 20th: Normal temperature and normal pulse (98.4° to 99.2° F., pulse 75), eruption becomes paler and has spread to the lower leg, also upon the dorsum of the foot, in the latter region consisting of small, irregular, partly net-like, arrangement of macules.

May 21st: The clinical history shows that the mucous membrane of the pharynx is

still slightly reddened and that the patient complains slightly of pain in deglutition. In the evening a temperature rise to 100.8° F. In the face distinct, small-scaled desquamation.

May 23d: As the remainder of the eruption, only a delicate marbling of the skin is seen, but the pharyngeal mucous membrane is still red, however, without swelling. The cervical lymphatic glands upon the left side are still markedly enlarged, those upon the right to a less extent. Splenic dulness amounts to  $5 \times 7$  cm. The patient feels perfectly well, has a good appetite, sleeps well and leaves the clinic on May 26th.

This eruptive disease has then shown itself to be an exceedingly mild almost afebrile affection. If the eruption, which was noted by the patient, had not appeared he probably would not have come to the hospital.

Among the *clinical symptoms*, apart from the description of the eruption, I shall especially mention *two* which are of some importance in röteln. These are: First, the *enlargement of the superficial cervical lymphatics* behind the sternocleido mastoid; and, second, the *enlargement of the spleen*.

Regarding the lymph glands, in our case the inguinal glands were also found somewhat enlarged. Very probably this glandular enlargement is less common, but those mentioned belong to areas of the body in which enlargement of the lymph glands may be most readily determined. In many cases this may also be noted in the axillary and cubital regions, or even in the small glands under the skin, upon the lateral aspect of the thorax.

This *lymph-gland enlargement*, which is never very considerable, but still quite plain, has a certain differentio-diagnostic importance according to the investigations made in this clinic.

In *measles* a lymphatic enlargement does not occur so early, although an enlargement of the lymph glands occurs after the development of the other symptoms, above all, depending upon the severity of the catarrhal phenomena of the upper air-passages. In *scarlatina* it is not primarily the superficially situated lymph glands, those situated behind the sternocleido mastoid, but those at the angle of the lower jaw and situated in front of the last-named muscle which, in connection with the severe inflammatory condition of the throat, enlarge early and are often even sensitive to pressure.

From this early, usually not considerable, enlargement of the lymph glands, there is to be distinguished a condition that occurs frequently in scarlatina, more rarely in measles, and perhaps also, occasionally, in röteln, a subsequent inflammatory swelling which often terminates in abscess formation. In scarlatina the condition not infrequently shows itself in the third and even in the fourth week after the onset of the disease, after the fever which is in connection with the process has run its course, developing a fresh febrile curve and occasionally arising simultaneously with the phenomena of scarlatinal nephritis.

The lymphatic enlargement in röteln, according to the observations which Clement Dukes made in a school, may be the first symptom of the disease, appearing before the cutaneous eruption.

The *enlargement of the spleen* is but very moderate, but, nevertheless, it is present from the onset of the affection up to the time of convalescence, and, if the contour of the spleen is drawn upon the skin and measured, it cannot be missed. The enlargement is somewhat less than in the case of scarlatina but more marked than in the case of measles in which the enlargement of the spleen, according to our observations, does not go beyond its usual borders.



Diagnostically, this may be of importance in doubtful cases as in all acute eruptive diseases the consideration of all the accompanying phenomena, as well as of the eruption, is of the greatest importance. The diagnosis in an acute eruptive disease, especially in childhood, may, under some circumstances, be exceedingly difficult and is often combined with great responsibility on the part of the physician.

Compared with measles, and especially with scarlatina, rōtheln is a disease of but very slight importance. According to the very careful investigations of Thomas, Emminghaus, and others, the disease runs its course entirely without fever, in some cases fever with a temperature of 102.2° F. It is highest at the onset of the disease and falls by rapid lysis. Therefore, in the case of rōtheln the fever is not in close connection with the eruption, as is the case in measles, in which the maximum of the acme of the fever is in proportion to the height of the eruption, and, then, if no complications occur to cause a deviation from its usual course, falls by crisis.

In rarer cases of rubella, besides a marked rise in temperature, there may be symptoms which are common to other febrile diseases, being in part dependent upon the individuality of the patient, such as general lassitude, headache and restlessness. Herpes labialis has also been occasionally observed.

Some *sequelæ*, such as eczema, herpes zoster, marked angina tonsillaris, purulent inflammation of the lymph glands, erysipelas, albuminuria, which have here and there been observed, are in no direct connection with the specific rubella infection, which, for the most part, is only the impetus for other infections to appear. These sequelæ, however, are very exceptional. It is the rule that rōtheln is a quite harmless affection, running its course, in many cases, without serious consequences. Therefore, when the diagnosis can be made with certainty from the first appearance of the eruption, although we are dealing with a contagious disease, to which the susceptibility in infancy is a very great one, isolation of the patient is not necessary. On the other hand, in a mild case of scarlet fever, in which we are never quite certain whether serious complications may arise or not, and from which, by transmitting contagion to another, the severest, absolutely fatal form of scarlatina may arise, isolation is imperative. In measles, at least as regards sickly or weak individuals in the cold seasons of the year, in which severe complications are likely, especially on the part of the lungs, isolation is advisable. There are, however, especially in scarlatina, mild forms with but feebly characterized, or but sparse and locally limited eruptions and slight throat phenomena. On account of the mildness of the affection, and perhaps also on account of the variety of the eruption, the danger of diagnosing rōtheln in such a case is especially great if, at the same time, rōtheln is epidemic, but, as is common in large cities, sporadic cases of scarlatina occur from time to time even without an epidemic. Suppose such a case occurs in a child belonging to a family in which there are many children, or happens in a house in which there are many families with numerous children, or in a large boarding school; rōtheln is diagnosed, the case is looked upon as mild and without serious conse-

quences, isolation is not regarded as necessary, and after the child appears to have recovered he is permitted to return to school. After a few days a brother or a schoolmate is attacked by typical scarlatina and perhaps succumbs rapidly to the severity of the affection or perhaps another child that also came in contact with an apparent rubella patient is affected by mild scarlatina, in the course of which, however, otitis media occurs with a permanent injury to the ear, or an endocarditis develops which gives rise to chronic valvular disease, or a severe renal inflammation, with uremic phenomena and dropsy appears, from which permanent changes in the kidney remain. Apart from the misfortune to the affected family, what consequences an occurrence of this kind will have for the reputation of the physician need not be enlarged upon. And all this might have been prevented if, by precaution, a positive diagnosis had not been made in the first place, but the patient had been isolated and the further course of the affection observed. What has been said is sufficient to call attention to the responsibility of the physician in cases of this kind.

From this it may be seen that in the diagnosis of eruptive diseases, especially in infancy, it is the duty of the physician to be exceedingly careful to take into consideration all factors and rather err on the side of too much care than, without certain signs of support, to make a positive diagnosis which subsequently proves to be erroneous. Every case that is at all doubtful is to be isolated if there is danger of the presence of a serious disease such as scarlatina or measles or variola, for these must all be taken into consideration in the first stages of the eruption.

**Eruption.**—The eruption in r  theln must be carefully considered as this is the symptom which impresses the stamp upon the affection, and it is the exanthem which is the most important differentio-diagnostic point in the eruptive diseases in general. Before describing the eruption of r  theln, I must state a fact which should be remembered in every case. It is this, that a cutaneous eruption, perhaps due to a specific cause of an infectious nature, is not alone dependent upon this cause for its appearance, but also upon the structure and condition of the skin of the affected individual, therefore, it is dependent also upon age, general nutritive conditions, circulatory circumstances, the manner of reaction of the nervous system and upon cleanliness. Regarding the skin, it is a well-known fact that in persons of the same age, one may have a fine, and the other a coarse skin; in the former the cutaneous glands are scarcely visible, whereas in the latter they are quite prominent; in one the epidermis is so tender that the capillary net may present itself as a reddish surface, whereas in the other redness is scarcely noted, even in the majority of light complexioned individuals, in parts that are usually prominently red, such as the cheeks, although such individuals may be in the main entirely healthy and by no means anemic.

According to the condition of the skin, the same cause which produces an eruption which primarily causes a macular dilatation of the superficial capillaries of the skin, in which, especially the capillary circle, the mouths of which surround the cutaneous follicles, is more markedly filled with blood will

produce quite a varying picture in different persons. I take it for granted that the skin is well cared for and that the appearance and composition of the eruption is not partially concealed by dirt or by disturbances in the circulation and by general asthenic conditions of the organism.

In fact, it must be further noted in regard to the eruptive diseases in question, that the manner and way in which the causative agents act upon the skin are varying and that the variation is influenced by the severity of the affection and, above all, by the virulence of the causative agent of the disease.

Therefore, there may be several circumstances which, in the individual case, may prevent an eruption from appearing in a typical manner, so that it will be well nigh impossible to formulate a diagnosis, except by means of the other conditions: proof of contagion from a well characterized case, the existence of an epidemic, symptoms and prodromal phenomena of a distinct and characteristic kind.

It must be mentioned, above all, that there is no typical uniform eruption in rōtheln. Of the three acute eruptive diseases that have been mentioned, the eruption in rōtheln varies the most.

Whereas in the case of *measles* there are indented macules, often with a small papular formation due to the follicles of the skin, which stand out prominently with a more or less well defined bluish color, the intensity depending principally upon the severity and distribution of the accompanying catarrh of the respiratory organs, the cyanosis being due to this, the scarlatinal eruption consists of very minute closely intermingled pin-point marks which are found upon the uniformly injected, reddish, discolored skin.

The diffuse reddening is due to a very uniform dilatation of the entire cutaneous capillary net, which, however, is by no means due to a paralysis of the vasomotors. On the contrary, the smallest cutaneous arteries even show an abnormally increased irritability in a similar, but more marked, manner than that which occurs in every febrile reddening of the skin in the most varied febrile diseases, and, next to scarlatina, is most notable in the case of enteric fever. If we run the finger nail or the end of the percussion hammer very gently over the skin there appears very rapidly in a few seconds after the disappearance of the pallor due to the pressure, a broad line 1 to 2 millimetres greater in extent than that produced by the nail or other instrument upon the irritated area; thus designs and words may be drawn upon the skin, these only disappearing in from two to three minutes by a slow redilatation of the previously narrowed vessels.

This sort of an erythema of the skin, naturally, can only occur in rōtheln when the disease runs its course with fever as this is a condition which is seen preëminently in febrile vascular dilatation. In measles this occurs particularly in the stage of the initial fever, before the appearance of the characteristic eruption. In this diffuse vascular dilatation, the underlying cause is apparently a peculiar action of the pathogenic agent of the disease or the effect of certain toxins produced by it upon the vasomotor apparatus.

The rōtheln eruption consists of, at times larger, at other times, smaller, slightly elevated papulo-macules, similar to those of measles but lighter in color, not bluish but of a rose-red color. In some cases these may be very



small so that the eruption at first sight resembles scarlatina more than it does measles. Such fine punctiform areas as occur in scarlatina we never see in r  theln. But during the fading of the eruption a phenomenon may appear which is seen in scarlatina and not infrequently in measles, namely very minute punctiform hemorrhages which are situated close together; these are particularly noted in the delicate skin of the axillary region below the external half of the clavicles. They are of no importance in so far as no other severe symptoms are present which point to a hemorrhagic diathesis or to sepsis. The individual macules of which the r  theln eruption consists, for the most part have an irregular serrated limitation in some cases, and in some areas of the body are so far apart that the entire quite normal appearance of the skin which lies between them may be determined with certainty. The macules give the impression of being slightly raised, but they are not as coarse, as a rule, as those which occur in measles.

Upon the covered parts of the body and at those places at which sweating is apt to occur, sudamina appear, similar to the case in other eruptive diseases.

The distribution of the eruption occurs from the head and face downward. It may, however, especially in cases in which the eruption is but slight, appear first upon the chest and arms, very frequently, even profusely, upon the face, neck, trunk and arms; the eruption is less copious below the knee, and on the lower leg and on the feet but few individual macula can be determined.

Regarding the appearance of the eruption, great variations may occur. One point, however, requires exact observation because it is of great diagnostic importance: In r  theln, as also in measles, the region about the mouth (upper and lower lips, point of the nose) is often profusely covered by the eruption, whereas in scarlatina this region of the face is quite conspicuously free from eruption and often remains free from any erythema so that it is conspicuous by its pallor in contrast to the redness of the rest of the face.

Another point, which in a diagnostic respect deserves observation and which I intend to emphasize again, is this, that in the great majority of cases the eruption in rubella is the very first symptom of the disease, whereas in the case of measles the eruption appears only upon the fourth day, and in scarlatina upon the second day after symptoms have appeared. In the case of measles these prodromal symptoms are: fever with coryza and lachrymation, and, according to the height of fever, more or less well developed general malaise, which upon the second and third day, with a decline in the fever, may increase. In scarlatina: very frequently at the onset of the affection, vomiting, fever and pain upon deglutition, with the signs of an "angina faucium"; with very intense reddening and a quite unusual increase in the pulse rate, even out of proportion to the high temperature.

Desquamation in a profuse r  theln eruption occurs in the form of small flakes, especially upon the face, neck and chest; it is, however, commonly overlooked if special attention is not paid to it.

If fever is present, the urine may transitorily show traces of albumin.

Several times in our hospital, in cases of rötheln, we have found the diazo-reaction.

Convalescence in this very mild affection is exceedingly rapid. Remarkable is the reappearance of the eruption after the complete disappearance of the first attack. But these very rare relapses of the disease, as Emminghaus has observed, even with a very rapid course, have their analogy in the relapses of other infectious diseases. Accurate descriptions will be found in the exhaustive monograph on rötheln by Johannes Seitz<sup>1</sup> in Zürich.

That in the case of rötheln we are dealing with a substantive, specific affection, sharply defined from measles and scarlatina, is now generally accepted. The reasons for the separation of both previously mentioned diseases, besides those already enumerated in the description of the symptoms, are these:

1. That rötheln, measles and scarlatina may follow or precede one another in the same individual, that, therefore, neither is a protection from rötheln, nor the latter a protection from either of the other affections, whereas a second attack may occur in measles, as also in scarlatina, but is, however, quite rare;

2. That the period of incubation is a different one from that in the case of the two other affections, in case of rötheln it is about three weeks, seventeen to twenty-one days, in measles only about nine days, and in scarlatina, under some circumstances, only one day, but may also be much longer. The contagious principle of scarlet fever is extraordinarily resistant so that by adhering to clothes and all possible substances (fomites) the transmission is often an indirect one. Between the adherence of the contagious principle to clothes, etc., and its entrance into the body which is still dependent upon all possible eventualities, a varying period of time elapses.

The great variations which the eruption of rötheln shows in different cases, so that it resembles measles, at another time scarlatina, are conditions which increase the difficulties of diagnosis provided the other symptoms and other circumstances are not sufficient to form an opinion. But a new diagnostic difficulty must yet be mentioned.

**The Fourth Disease.**—A few years ago, while physician to the great boys' school at Rugby in England, thus having an unusually large field of observation for these diseases, Clement Dukes observed that in children that have gone through an attack of rötheln an eruptive disease may appear in which the eruption more closely resembles scarlatina, the other symptoms, however, being against the assumption of scarlatina. If we are dealing with relapses from rötheln it is conspicuous that in the second attack the eruption is quite different from that of the first attack. Dukes suggests that there is possibly still a fourth disease which has something in common with scarlatina and with rötheln, but which is neither one nor the other affection. Other physicians also, principally American and English observers, have, in the course

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<sup>1</sup> *Correspondenzblatt für Schweizer Aerzte*, Jahrg. xx, 1890.

of the last seven years, published observations which are calculated to confirm the assumption of a fourth disease ("fourth disease" as Clement Dukes<sup>1</sup> has named the affection). According to Weaver,<sup>2</sup> the symptoms are, an eruption resembling scarlatina but which also occurs around the mouth, very slight fever, no increase in the pulse rate, no vomiting, no, or but very slight, throat phenomena, no desquamation as in the case of scarlatina, and an absence of the lymphatic gland enlargement of r  theln. F. T. Simpson<sup>3</sup> noted the affection several times after scarlatina, in numbers of instances after measles. He observed in several patients of this kind lamellar desquamation.

**Diagnosis.**—In children, with their much more susceptible skin and irritable nervous system as compared with adults, there occur, under the influence of the most varied causes, more or less well developed eruptions which, at least in their primary stages, may cause confusion with one or the other of the specific eruptive diseases. Especially in small children, during the period of dentition and in seasons in which intestinal catarrh is frequent, with or without febrile phenomena, acute eruptions of brief duration occur, which have been called "roseola aestiva" and which may resemble r  theln. In such cases r  theln can only be diagnosticated if, at the same time a r  theln epidemic is present, contagion can be proven, or if from a patient of this sort contagion is spread and an affection resembling rubella appears. In such cases lymphatic enlargement and enlargement of the spleen must be present. Many affections of this kind belong to urticaria or also to erythema multiforme, which latter affection may also run its course with lymphatic enlargement and enlargement of the spleen.

Etiologically, intoxication or autointoxication plays a prominent r  le in these cutaneous eruptions; in erythema multiforme, probably an infection is the cause. It is sufficient to mention the urticaria produced by the eating of strawberries, by the ingestion of certain kinds of sea-food, that form produced by intestinal worms and by digestive disturbances of various kinds, which, in some persons, especially in early youth, is likely to produce this form of eruption. As great as is the difference between a macule of r  theln and of even a small patch of urticaria, it must not be forgotten that, especially in children, any eruption, by friction of the clothes or of the bed and above all, by scratching, the irritated areas may develop papules, even wheals. It must be further remembered that a purely local irritation of the skin, in individuals that are especially susceptible, may cause a distribution over larger areas of the skin, by means of scratching; these primarily local irritations being caused by the bites of insects, by the hairs of certain caterpillars, by ethereal, oleaginous and other vegetable products (thuja—and pinus varieties, primulacea).

Many drugs may, in some individuals, children as well as adults, give rise to eruptions which may distribute themselves over the entire body. On account of its great similarity to measles, the first to be mentioned is the eruption

<sup>1</sup> *Lancet*, July 14, 1900.

<sup>2</sup> *Dublin Journal of Med. Science*, 1901, VI. Ref. *Fortschritte der Med.*, 1901, Nr. 35.

<sup>3</sup> *Archives of Pediatrics*, Sept., 1901. Ref. in *Am. Journ. of Med. Sci.*, Jan., 1902.



caused by antipyrin, which, in children that have had antipyrin administered to them on account of whooping cough, is not so infrequently observed. In comparison to the eruption of rōtheln, the latter is of a darker color and of a more papular character.

The eruption appearing after the injection of diphtheria antitoxin serum may consist of flat macules or papules, and, if at the same time an epidemic of rōtheln is present, may give rise to confusion. This eruption, as a rule, shows larger macules than occur in rōtheln, usually irregularly distributed over the body and, therefore, more closely resembles erythema multiforme.

In some persons there is a remarkable idiosyncrasy to quinin, in others to mercury, upon internal and external administration. In such persons even minimum doses are sufficient to cause a dermatitis which distributes itself over the entire body, in which only at the onset the erythema is small, macular, soon becoming uniform so that the eruption of scarlatina is closely simulated. This diffuse dermatitis, running its course with fever, is accompanied by a desquamation of the entire epidermis, large flakes coming away, and occasionally, also, by a falling out of the hair and the nails.

In adults, occasionally, the internal administration of cubebs or balsam copaiba is the cause of a small papular eruption which may resemble measles or urticaria.

In some regions in which typhus fever is endemic, or where it is brought in, measles may be thought of on account of a very profuse eruption, a diffusely livid discoloration of the skin and the papular consistence of individual florescences. These papules, however, are only a small constituent of the eruption, which, in the main, consists of entirely flat, irregularly constituted, livid areas, frequently intermingled with petechia—hence the name petechial typhus. The entire course of the disease, in which the eruption appears only upon the fourth day, the high fever, the marked enlargement of the spleen, the occurrence of typhus fever in the affected region, and the probability that contagion has taken place, enable us, with some slight care, even when measles and rōtheln are simultaneously epidemic, to avoid an error, which, on account of the greater transmissibility of typhus fever, might give rise to serious consequences.

In epidemic cerebrospinal meningitis, in some few cases eruptions occur which show a certain similarity to measles or rōtheln, as a rule, however, they are more localized to the extremities, a part of the trunk, and more closely resemble erythema multiforme than the previously mentioned eruptive diseases. The presence of an epidemic and of undoubted cerebrospinal inflammatory phenomena is determining above all in the differential diagnosis.

In an adult, during the prevalence of an epidemic of rōtheln, a very profuse eruption of roseola syphilitica may appear during the eruptive period of this disease, possibly at the same time with the febrile phenomena, so that the true nature of the disease is concealed and rōtheln is thought of. In both, enlargement of the lymphatics and of the spleen is present, in both there may be a somewhat more decided erythematous condition of the palate with

slight swelling. But in an eruption appearing so acutely, especially with fever, there is a much more decided general malaise, and, above all, as a rule, severe headache which increases toward night, and the greater gravity of the disease is already noted in the facial expression. Even to the inexperienced, the continuance of the eruption beyond three to four days, its increase, not from above downward, but a closer approximation, and an increased development of macules into papules will soon show the true nature of the affection. The examination of all palpable lymph-gland areas will then show at some place in the body a few that are more markedly enlarged, being painless round glands ("indolent buboes"), and, in the peripheral area of the same, the primary lesion may be discovered.

Finally, in septic affections, eruptions which develop rapidly, being of a macular or papular character, not infrequently resembling urticaria, occasionally being hemorrhagic or postulous, have, at certain stages, some similarity to r $\ddot{o}$ theln or even measles. By an observation of the accompanying phenomena errors may easily be avoided.

It will, therefore, be seen that in the individual case many possibilities must be thought of before an opinion is given in an acute eruptive disease. Above all, every eruption should be accurately examined, and whenever possible, by daylight, and the most recent efflorescences should be sought in order to determine their original form, and the further development of the eruption should be compared with these. The opinion should never be made to depend upon the appearance of the eruption alone, and especially not in those cases in which the affection appears to deviate from the ordinary type of the disease. Above all, the prodromal phenomena of the disease should be inquired into, those that were present before the appearance of the eruption. A complete absence of these, as has already been noted, occurs especially in r $\ddot{o}$ theln, whereas in measles a prodromal stage lasting three days, and in scarlatina, one to two days, is present, accompanied with fever, in measles with catarrh, primarily of the upper respiratory passages, in scarlatina with angina faucium, vomiting and an unusual increase in the pulse rate. But very rarely is there a constitutional disturbance or sore throat, or coryza in r $\ddot{o}$ theln. In measles, before the appearance of the eruption, an alteration of the mucous membrane of the cheeks occurs, which is not observed in the case of scarlatina and r $\ddot{o}$ theln and for this reason is of great diagnostic importance. These are the so-called Koplik's spots: isolated red points the size of the head of a pin with a bluish-white or whitish-yellow point in the centre. In every case in which measles is suspected these should be searched for. After the appearance of the eruption they become indistinct or disappear completely, probably under the influence of a more marked hyperemia of the mucous membrane.

Then the other organs (lungs, heart), above all, the spleen and lymph-glands are to be examined; in an examination of the urine the diazo-reaction should not be forgotten.

A careful investigation of the accompanying circumstances, the possibility of contagion within the period which corresponds to the incubation period of

the disease should never be neglected, especially in cases in which there has been no previous appearance of the disease in the family to clear the situation.

In every doubtful case a positive diagnosis should not be made, but care should be taken that the disease is not transmitted to others, the patient should be isolated and the diagnostic decision should be deferred for the next few days, until the disease has developed fully. As regards *the treatment* of the patient, nothing will be neglected by this. In those cases in which special symptoms are prominent they must be treated on general principles. High fever and cerebral symptoms in scarlatina and measles may require cold baths, in röteln symptoms which require treatment almost never occur. In a case of röteln, therefore, general dietetic hygienic rules are to be followed which are self-evident in any mild affection.

**Differential Diagnosis.**—A table is appended which shows the characteristic differences of the three diseases at a glance:

	RÖTHELN	MEASLES	SCARLATINA
I. INCUBATION PERIOD.	17–21 days.	9–11 days.	1–7 days and longer.
II. PRODROMAL SYMPTOMS BEFORE THE APPEARANCE OF THE ERUPTION.	None.	Fever, catarrh of the upper respiratory passages and the conjunctiva.	Vomiting, sore throat, fever, increased rapidity of the pulse.
III. ERUPTION: First appearance. Distribution.	First day.  First in the face.	Fourth day.  The same.	Second day.  First upon the neck and upper parts of the chest, the face, the region about the mouth remaining free.
Character of the eruption.	Macular or punctiform, color light red.	Macular, slightly raised, prominence of the cutaneous follicles, color bluish red.	Finest punctiform areas, close together upon a uniformly reddened skin. Color deep red.
Desquamation.	Ill-defined, small flakes.	Small flakes.	Upon the neck and trunk small flakes, upon the hands and feet, often only after weeks, in the form of large scales (lamellous).
IV. ACCOMPANYING SYMPTOMS: Throat.	Catarrhal reddening.	At the onset, macular reddening then uniform (upon the mucous membrane of the throat, Koplik's spots).	Uniform marked inflammation with more or less swelling, occasionally follicular plugs or membranous deposits. Complicated by diphtheria.



	RÖTHELN	MEASLES	SCARLATINA
IV. <i>Continued</i> Respiratory organs.	Sometimes catarrh.	Marked catarrh, laryngitis, bronchitis and bronchiolitis. Great tendency to broncho-pneumonia.	
Spleen.	Some enlargement.	No enlargement.	Enlargement.
Lymphatics.	Superficial cervical, auricular, often also other glands, soft and frequently sensitive to the touch.	Not so constantly enlarged as in rōtheln.	Glands, especially at the angle of the lower jaw enlarged; cervical glands later on, as a complication often with abscess formation.
V. FEVER.	Often no fever, or but slight rises in temperature, rarely to 102° F. Highest temperature usually upon the first day.	Prodromal fever lasting three days. Temperature rises with appearance of eruption, maximum of both at the same time.	Highest temperature at the onset.
Defervescence.	Lysis.	Crisis.	Lysis.
VI. COMPLICATIONS AND SEQUELS.	No constant nor frequent ones. <sup>1</sup>	Broncho-pneumonia, otitis media. Tuberculosis of bronchial glands and lungs. General military tuberculosis. Noma. Diphtheria. Rarely nephritis.	Endocarditis. Acute rheumatism. Nephritis. Otitis media. Septicopyemia.

<sup>1</sup> The complications and sequelæ mentioned under measles and scarlatina are not "constant." On the other hand, the presence of nephritis does not exclude the diagnosis of "Rōtheln." It will occasionally occur as a sequela.—EDITOR.

# MEASLES, MORBILLI, RUBEOLA

BY O. HEUBNER, BERLIN

**Definition.**—By the designation measles, we understand a febrile disease of the upper respiratory mucous membranes and of the general cutaneous coverings, having quite a typical course in its symptoms, belonging to the acute exanthemata, and which is always due to the entrance of a specific poison derived from a previous case of measles.

**Etiology.**—Although in the descriptions of authors of the middle ages measles cannot be recognized with certainty, it is at least likely that we are dealing with a very ancient disease, perhaps of the same age as variola. The susceptibility of the human race to the poison of measles, in spite of repeated infection, has not been attenuated even to-day, for but very few persons, comparatively, fail to have an attack of measles when they come in contact with a patient ill of the disease. Even the fetus in utero may be attacked by measles by means of the maternal blood, and the aged also are subject to the disease if they have never been previously exposed to the contagion. This has been noted in members of royal families or inhabitants of islands, in which for decades contagion has not appeared. On the other hand, no one is ever attacked by measles who has not in some manner or other come in contact with a case of measles, in Iceland as well as in Central Africa, i. e., in other words: Only a specific poison developing anew in man is capable of producing the disease.

This poison only develops in man and not outside of the human organism, for in his surroundings it does not usually remain active very long; for even a residence in places in which measles patients have been previously present, a few hours after careful ventilation, confers no danger to susceptible individuals. It may be that the poison, by means of directly infected body linen, utensils, perhaps also, for example, by a letter<sup>1</sup> that has been rapidly closed, may be transported to certain distances inside of a short period of time, in this way bringing about a contagion; but without question this is not possible after a prolonged period. Its increase in the affected organism is very decided, for a single patient is capable of conveying the disease to dozens of susceptible individuals, and by a further development hundreds and thousands of human beings may be infected, as has occurred upon isolated islands (Faroe

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<sup>1</sup> A well-authenticated case was described in *Brit. Med. Journal*, June 22, 1907, by Hughes R. Davis.—EDITOR.

Islands) twice in one century, the condition having been observed by careful and responsible physicians.

With the exception of individual rare cases which appear to be endowed with a congenital personal immunity against the disease, so that in spite of frequent opportunity of contagion they are never affected, there is but one circumstance which conveys protection from the poison, that is recovery from the disease. The immunity acquired in this way in most persons is usually permanent, it commonly lasts for the entire life. Unquestionably measles has sometimes occurred several times in the same individual, but this belongs to the greatest raritiës and does not vitiate the rule. I remember having seen two attacks of measles (in an interval of several years; relapses are not meant) in a child in which the first attack was only rudimentary. We often hear, in the treatment of a case of measles, that the patient has already had an attack of the disease.

This permanent immunity is all the more remarkable as another immunity artificially acquired, that against diphtheria, in no other acute affection appears to disappear so rapidly as in the case of measles.<sup>1</sup>

The region to which the poison first adheres in the body of the affected individual, how it first distributes itself, in what connection it occurs with the symptoms of the disease, are up to the present still unknown facts. This may be readily explained as we have absolutely no knowledge regarding the origin and nature of the contagion. All endeavors of the best investigators to lift the veil of this secret have remained without result. If we may conclude from other analogies, the primary point of attack of the virus might properly be referred to the upper respiratory mucous membranes. It has not as yet been determined by conclusive experiments, by means of what secretions or excretions of the body the contagious principle is transmitted to the healthy. In the case of measles we usually speak of a fleeting contagion, but observations like those of Grancher do not favor the view of the immediate transmission of pathogenic bacteria from the sick body by means of the air. This investigator, in a ward in his hospital for children, by a process invented by him (wire boxes around every bed) prevented the individual patients from coming into direct personal contact with a measles patient, whereas, naturally, the current of air from one bed to another was not hindered. When a child attacked by measles was brought into the ward at the onset of the period of incubation, it was shown that not the neighboring children were attacked, but a child was affected which was attended by the same nurse as the child that had the attack of measles. This nurse had nothing to do with the other children because both the mentioned ones had been nursed while isolated on account of suffering from another infectious disease (scarlatina). In the usual course of things, naturally, those nearer the measles patient are more threat-

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<sup>1</sup> Many of our readers may have had a different experience. Measles does not create such an immunity as is here claimed. It may reappear several times in the life of a child. Immunity from diphtheria is not caused by a single attack; on the contrary, repeated attacks spread over years are not uncommon.—EDITOR.



ened than those distant, but even here the transmission occurs more by contact (touch, transmission of infected substances and the like) than by the air. Naturally, upon close proximity the transmission by means of vaporous particles (in sneezing, coughing and the like) is important.

After reception of the poison by the healthy, a number of days pass before the first symptoms of the disease appear. This time, called the *period of incubation* of the disease, is in most individuals, in the case of measles, a regular period of about eleven days. This was first determined by Parnum on the Faroe Islands where there was frequent opportunity of studying the course in such patients as exposed themselves but a single time to the contagion. The exanthem usually appears exactly upon the fourteenth day after contagion has taken place. Later, when it was possible, by means of taking the temperature, to note the first onset of the præruptive period, it was shown that the period of incubation in a restricted sense, required the previously mentioned eleven days.

The general susceptibility to contagion shows a certain limitation during the first four or five months of life; but even here there is no absolute immunity. I observed an undoubted attack of measles with very characteristic symptoms, with fever and well-developed eruption in a boy aged fifteen weeks, whose brother and sister, aged respectively three and one and a half years, simultaneously suffered from extraordinarily severe and rapidly fatal attacks of measles; the sister died four days after, and the brother three days after the appearance of the disease in the nursling. In a second case I saw a boy aged twenty weeks attacked by measles, with a very marked and intense eruption, with high fever. In both cases rapid recovery without complications occurred.

After the first year of life the disease is much more frequent, and from the second year on the susceptibility is the same as later in life.

The susceptibility which is quite general, on the one hand, and the immunity acquired by an attack, on the other hand, determine the character of measles as an *epidemic* disease. It is only an apparent disease of childhood, as, almost without exception, in all larger areas of population all individuals have had an attack of measles before they have passed beyond the limits of adolescence. Where measles breaks out in a population in which no immunity has been acquired by previous attacks, as in 1846 upon the Faroe Islands, every one is attacked. Among nearly 8,000 inhabitants upon the island only those old persons were spared who sixty years previously, while they were children, had passed through an attack of measles.

The seasons of the year have only an influence in so far as there is a connection between a general closing of schools and kindergartens, as during vacation, and in other institutions for children. From this concentration of young children, the occasional upflaring of measles in large cities always renews itself if, since the last larger epidemic, a greater number of children who are not yet immune have grown up, so that they find entrance into the previously mentioned institutions, in which there is always recurring oppor-

tunity for contagion through some child attacked with measles coming from an infected district of the city. Thus, as Medical Director of a district, I formerly had an opportunity in April, May and June as well as in October and November, to observe marked epidemics of measles. The older children bring measles home from school to the younger members of the family, and where a population, particularly in case of the poor, live close together in the same street, the disease spreads rapidly from one house to another and from floor to floor in the same house, until many children recovering from the malady have become immune to the disease, the locality thereafter showing no cases for several years. The same conditions prevail in smaller cities and villages in the case of all the children in certain districts.

The same phenomenon is noted in the wards of a children's hospital unless it is especially protected. A child is admitted on account of catarrhal affection which is not recognizable as belonging to measles and on the eleventh day an epidemic occurs in the ward, attacking all children that have not been protected by a previous attack. Here the results are much more serious than in the city, for in the hospital the affection attacks children that are already ill from other causes, producing *secondary* measles, the disease then being far more serious.

### PATHOLOGICAL ANATOMY

The anatomical examination of children that have died of measles does not give the slightest clue regarding the nature of the disease. The parts of the body and the organs which during life have shown distinct deviations in form and color as well as in function at the autopsy show no alterations which characteristically differ from catarrhal and inflammatory phenomena in the same parts in a number of other affections. In the skin the dilatation and tense congestion of the small veins and capillary nets of the cutis are recognized, which may be assumed from the clinical symptoms. Along the walls of the smallest veins leukocytes are found arranged in rows, the lymph channels are wider, the skin succulent, and disseminated foci-like collections of round cells are present in many areas of the cutis. The entire inflammatory or vaso-motor change of the skin in measles appears particularly to attack the upper layers nearest the epidermis in which cutaneous vascular nets are present. The hair follicles and sebaceous glands are surrounded by great numbers of capillaries; the infiltration of these structures is probably the cause of the nodular prominences which characterize the eruption of measles.

The catarrhal process in the respiratory mucous membrane perhaps shows anatomically a somewhat greater intensity in that the vascular system of the submucosa is more markedly implicated and the extra-vasal infiltration of the mucous membrane is greater than in ordinary catarrhal processes. This is of especial importance in regard to the course of the catarrh of the ears and of the larynx. When the catarrhal affection enters the finer and finest bronchi

there is an early development of interstitial cellular infiltration of the peribronchial and perivascular connective tissue.

Almost always—especially in the severer rapidly fatal cases—(as in many acute infections of infancy) a very decided cellular hyperplasia of the entire lymphatic system develops. All peripheral and internal lymph glands and other conglomerations of adenoid substance such as the pharyngeal and palatine tonsils, the solitary follicles at the root of the tongue, walls of the pharynx, and throughout the entire intestinal tract such as Peyer's patches of the ileum, are found to be in a condition of intense medullary swelling, so that an inexperienced observer not infrequently, especially upon viewing the lower parts of the small intestine, erroneously supposes enteric fever to be present. The acute hyperplasia of the spleen in measles is as a rule less developed.

Whether this marked cell infection of the lymphatic organs which occurs in a few days is in connection with the regular appearance of leukocytosis in measles has not yet been determined with certainty. [In uncomplicated measles leukocytosis does not occur.—ED.] At all events, in the child, even in cases in which the number of leukocytes is not markedly increased, the blood picture is altered in the manner that the lymphocytes are decidedly less than the polynuclear leukocytes. Large forms of these, such as mast cells, some authors have met with more frequently in the blood of measles than elsewhere.

The heart occasionally shows implication from the poison of measles, especially the endocardium is not rarely involved, whereas the pericardium is not so liable to become affected. The myocardium may also occasionally be damaged by the poison.

The vascular twigs and the parenchyma of the kidneys are less frequently damaged than in the case of scarlatina, however, occasionally the renal structure is attacked and then shows conditions that occur in scarlatina.

The pathological alterations of the lungs will be considered in describing the deviations from the normal course of the disease.

### CLINICAL PICTURE OF MEASLES

In measles it is practical to differentiate various phases or stages of the clinical course which may be distinctly recognized at the bedside.

After the stage of incubation which was explained above, the *catarrhal stage* follows, this is succeeded by the *eruptive stage*, and this, again, by the *period of convalescence*. The catarrhal stage is also designated as the stage of invasion, and, formerly, the name prodromal stage was given to it. The stage of eruption is spoken of as the stage of bloom or florition.

These individual periods often find a very characteristic expression in the picture of the temperature curve. Thus, in one of the following charts, that of a boy aged twenty-one months, the fever, which accompanied the mucous membrane affection in the catarrhal stage, showed a curve of three



days with two remissions; from the fourth to the seventh day a continued fever followed during which the exanthem appeared over the entire body, and after complete development of a cutaneous eruption the temperature fell in a critical manner, entering upon the stage of convalescence. (Compare the two following temperature curves, Figs. 20 and 21.)

The character of the fever—remittent in the first days, highest and most continued during the development of the eruption and falling rapidly after the complete development of the eruption—is noted in all typical cases of the disease and, even in regard to decisive exacerbation and remission in the various phases of the course as well as in regard to the absolute height of the fever, the greatest variations occur. That there is a connection between the highest temperature and the greatest intensity of the eruption, as was previously mentioned, can only be decided with certainty by one who has observed a great number of cases, as well in regard to the eruption as to the continued temperature course. This has not been carried out by any one as yet; and it scarcely appears to be of practical importance. But it is important to note whether or not the characteristic course of measles is shown; to observe this through the most manifold variations is hardly difficult to the trained clinical eye; to deny it means to disregard a well established fact in a very careless manner. I am of the firm opinion that some day the discovery of the poison of measles will clear up the clinical type of the affection in the same fortunate manner as in the case of malaria.

In connection with the fever we shall first describe the regular course of the affection.

The *period of incubation* of measles is usually free from symptoms, deviation from this rule will be described later on.

The onset of the disease is but little characterized and differs only slightly from an ordinary bronchitis. In daily practice the chronology of the whole course is much more difficult to determine than in the case of scarlatina or even diphtheria, because the parents are not able to say exactly when the "coryza" began. Only where—in a previous illness—the temperature of the child has been taken from the beginning of the disease (as in charts 20 and 21) is the rise in temperature above the normal noted from the onset (early in the morning in the rectum above 99.5° F., in the evening over 100.5° F.), even though the child very often shows no distinct subjective disturbances in its general condition.

In cases where this is absent there is no reason, even for very anxious parents, to send for a physician. This only occurs upon the appearance of an exanthem and thus it happens that the practitioner often enough does not have an opportunity of observing the first or the catarrhal stage of measles; and even in cases in which he takes regular records of temperature, he only notes the second half of the above chart as a fragment of the entire temperature course of the disease.

Where there are distinct symptoms on the part of the mucous membranes these consist of coryza, discharge from the nose, sneezing, swelling of the

nasal passages, disturbance of sleep; in small children shortness of breath, and occasionally severe epistaxis. To this is added—which is quite rare in ordinary coryza—a sense of pressure in the eyes, causing rubbing, injection of the conjunctivæ, lachrymation, and photophobia, so that the children ask to have the room darkened. The cough is often quite characteristic of measles; it is dry and paroxysmal and sounds harsh without any signs of

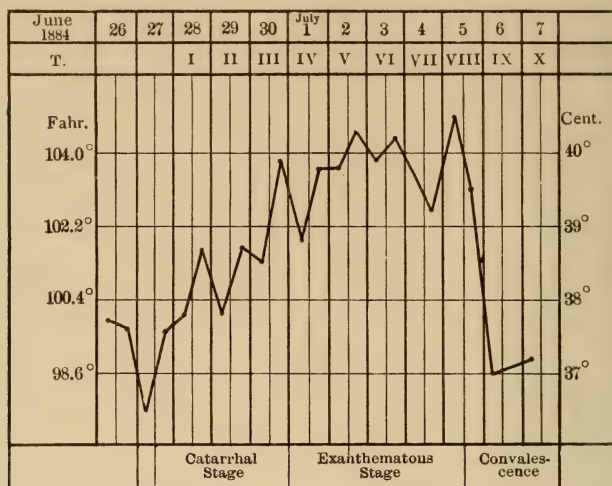


FIG. 20.—TEMPERATURE CHART IN MEASLES.

“looseness.” Finally, the voice is muffled or is hoarse from the onset, which may always be referred to a superficial catarrh of the larynx. Not always are all of these cardinal symptoms present, often only one or the other, and then only to a very slight extent. A possible disturbance of the general condition is shown in younger children by irritability and crying, in older ones by loss of appetite, lassitude, occasionally by chilliness or chills. Finally, not rarely, vomiting may occur as an initial symptom.

None of these complaints or difficulties has a well developed plastic character, but, rather, at least in the majority of cases, are only feebly developed.

The objective examination, if there be an opportunity to make one, does not show specific lesions in the diseased mucous membranes. The conjunctivæ are often intensely swollen and reddened, mucus and pus are excreted, and stick to the lids, thus causing difficulty in opening the eyes, but a macular character of the redness cannot be noted. If at all present, this is met with in the mucous membrane of the palatine arches and upon the tonsils, but only toward the end of the catarrhal stage shortly before the appearance of the eruption.

All the more valuable, therefore, is a phenomenon which, although not absolutely characteristic of measles (by some authors also noted in rōtheln), is of importance for the eruptive character of the catarrhal process of the

mucous membranes. This is the *whitish spots* found upon the inner surface of the cheeks, behind the angle of the mouth. This name may be given to them, as Koplik, who accurately described this symptom, states that they appear like fine specks of lime upon the reddened mucous membrane. These spots, found upon reddened areas, are about the size of the head of a pin, raised above the mucous membrane, disseminated, few or many in number, and of a bluish-white or yellowish-white appearance. Sometimes they can only be seen with a good oblique light, but if this area is examined they are often found to be quite prominent. Sometimes they resemble beginning aphthæ. These spots cannot be wiped away, and if the white deposits are scratched off they are shown to be collections of epithelium which are permeated by a turbid mass of detritus. They have previously been described, for example, by Reubold, a pupil of Rhimecker, by Filatow, and others, and they are also mentioned in Gerhard's text-book, but their diagnostic importance and their regular appearance on the *first* days of measles was first clearly shown by Koplik. They are a very frequent prodromal symptom, and in my clinic were noted in six-sevenths of all cases.

The symptoms of invasion last three days. Toward the end of this first stage, not infrequently before the cutaneous eruption, there appears upon the mucous membrane of the palate an exanthem which consists of individual, deep red serrated spots, separated from one another, inside of which the swollen solitary follicles are noted as individual nodules. This redness which

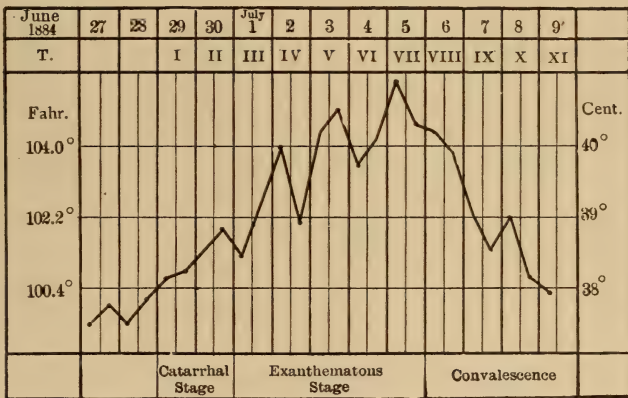


FIG. 21.—TEMPERATURE CHART IN MEASLES.

is sometimes confluent and only serrated at the borders occurs particularly in the velum of the palate, but thence distributes itself to some distance over the mucous membrane of the hard palate and but rarely, when quite distinct, affects the tonsils, for example, the surface surrounding the lacuna. This eruption on the palate disappears more rapidly than the cutaneous eruption. It is not of great importance for the diagnosis of measles to rec-



ognize this prior to the eruption of the cutaneous exanthem, as it usually only appears with this or at least precedes it only by a very brief period.

Upon the evening of the third day or during the succeeding night the first macules appear upon the skin, most frequently upon the face or about the eyes and mouth, upon the temples and behind the ears. But quite regular cases occur in which the first delicate macules are seen upon the back, whereas the head and face are still free from the eruption. The macules rapidly increase in number and enlarge by coalescence of the smaller and larger spots and now appear upon the entire surface of the body. A distinct sequence of individual parts of the skin may be noted, thus, after the head and neck, the upper trunk and upper arms, then the lower trunk, buttocks, thighs, and, finally, the forearms, lower legs and feet are attacked. The rapidity with which the entire body is covered varies greatly in individual cases, upon the average the maximum of the exanthem is reached in from a day and a half to two days. Upon its first appearance the individual macule of measles is small, scarcely of pin-head size, of a light red color, and round, sometimes, however, even upon its appearance it is of an irregular shape, oval, indented and also serrated. It is not raised above the level of the skin and in children with very white skins it is sharply defined. However, the character of this macule changes rapidly. It becomes darker red, dull brown, and larger, several neighboring macules being combined, and only now does the characteristic appearance of the eruption of measles appear: Lentil-sized, and larger, irregular, serrated, crescent-shaped macules inside of individual ones, in the larger macules mostly three or four conically pointed red millet-seed nodules are seen and felt. The nodules often correspond to hair follicles or to the mouths of sebaceous follicles. These glands secrete more profusely than normal so that the affected cutaneous areas have a slightly greasy feeling. The entire macule is now raised above the surface of the skin (*morbilli elevati*; cases in which this nodular elevation is slight or absent are known as *morbilli laeves*).

Occasionally in place of the nodules, particularly upon the back and chest, vesicles are formed, then a large number of small spots are found which carry a vesicle in their centre, the exanthem of measles then resembling *miliaria*.

The whole appearance of the measles patient shows distinct macules at the height of the eruption, the entire skin from head to foot is covered with closely adjoining individual points of the eruption. As the eruption in each part of the body remains at its acme for quite a time the total distribution of measles may be easily observed. No part of the skin remains free, the hairy head and ears as well as the genitalia being covered with the eruption. The back of the hands and the soles of the feet are shown to be most markedly affected.

Upon the trunk and upon the buttocks most frequently—but this phenomenon also occurs upon the face and the extremities—the appearance of the eruption is also altered by the fact that sooner or later the individual serrated macules extend beyond their limit and completely coalesce, forming large

clusters. This gives these wide cutaneous surfaces an appearance of being uniformly covered by a connected eruption, the consistence differing quite markedly from the spotted skin of ordinary measles. *Confluent measles* is then spoken of. That these have, however, occurred secondarily from the above-mentioned characteristic macules can always be noted upon closer examination, for individual, completely white, usually somewhat serrated cutaneous areas have remained in the midst of this connected mass of redness.

An appearance which is not rare in a quite regular course of the disease consists in the fact that more or less distributed areas of the cutaneous eruption become hemorrhagic. This occurs particularly upon the cheeks, forearms and buttocks, but it may also occur upon any other part of the body. That the vascular change in the macules of measles is combined with a greater permeability of the vessel walls permitting the hemoglobin to pass out (whether this be in the form of the erythrocytes themselves or in the form of dissolved hemoglobin from the corpuscles) may be concluded from the pigmentation of the macules of measles, which for days and even for weeks permit us to still recognize the character of the eruption which has already faded. Not rarely do massive extravasations of blood occur in the area of the eruption. Then the exanthem takes on a dark bluish-red color which gradually turns green or yellow, exactly like any other cutaneous hemorrhage. The change also preserves the exanthem for a long period even into convalescence. This deviation which is without importance for prognosis must not be confounded with the cyanotic appearance of the cutaneous eruption which is always of serious import and which will be described later on.

It is unquestionable that the patient during the acme of the eruption emits a peculiar odor which is variously described by different observers.

With the development of the exanthem the *complete disease* reaches its acme. The temperature at its highest grade reaches 104° F. and often above 104° F., sometimes above 105.5° F., even in ordinary cases, and even during the morning hours of the worst days no remission occurs. In a regular course of the affection the pulse corresponds to the fever without reaching disquieting heights. The general condition and the nervous system are now markedly implicated in the process. The appetite is entirely gone, the patient becomes apathetic toward his surroundings and his own sensations, toward night somnolence changes to irritability; complaints of headache and pains in the limbs, and not rarely delirium, often of quite severe type, may be noted.

All catarrhal phenomena increase. The mucous membrane of the eyes secretes more markedly, and during the night the eyelids become agglutinated, and can only be opened with great pain and then marked photophobia is present. The nose secretes purulent mucus, which excoriates the nasal openings and the upper lip. Now and then herpes develops about the mouth. The hoarseness is of a high degree; soreness in the larynx or along the larger bronchi is disturbing, but the dry cough which is almost continuous is particularly troublesome to the little patient weakened by fever.

Physical examination shows the existence of a bronchitis of the larger tubes.

The tongue is covered by thick, whitish, viscous masses which during the acme of the disease are often desquamated in serrated flakes so that the smooth red mucous membrane of the tongue is visible beneath. Upon the gums and upon the mucous membrane white deposits appear while the exanthem upon the mucous membrane disappears. Occasionally the eruption upon the skin is accompanied by a paroxysmal diarrhea, as a rule, however, constipation is present.

All the lymph glands of the neck, throat, axilla and inguinal region often enlarge decidedly, the glands may even become painful.

The urine becomes scant, concentrated, shows a sediment and, with a high fever, not rarely contains albumin. Various finer alterations in its composition point to disturbances of metabolism at the height of the disease; thus, the regularly and very markedly developed *diazo-reaction* which occurs in measles, the appearance of copious amounts of *diacetic acid*, of *propeptones*, allow of the conclusion that under the influence of the poison of measles the decomposition of albumin bodies of the organism occurs in a different manner than under normal circumstances.

Of the alterations in the morphology of the blood, mention has already been made; they point to the circulation of foreign toxic substances. That the blood during measles becomes rich in measles antitoxin, Weissbecker believes to have proven by experiments, in having cured severe cases of measles by the inoculation of serum from persons having recovered from that affection. An interrogation point may well be placed after this statement.

The description given above corresponds to the picture of the well developed intense but uncomplicated affection. In individual cases, in general practice, the accompanying phenomena are often much less developed even in instances in which the eruption, as is often the case, is well marked.

In the majority of cases, it is, however, characteristic that the disease develops progressively up to the maximum of the eruption so that the severest period corresponds about to the time at which the exanthem is in full bloom.

From point to point the combat between the organism and the poison of the disease takes place. The areas involved become more numerous, the condition is more and more threatening until a halt occurs which takes place rapidly and suddenly in the measles process.

During the same night in which the most severe delirium is present, in which the troublesome cough disturbs sleep, the swelling of the eyes and nose gives rise to the greatest discomfort and when the fever is almost unbearable, the entire picture changes suddenly; fever falls, the child sleeps, the cough becomes looser and sweating appears. Upon the next morning the child is without fever, and has a clear mind, appetite begins to reappear and he is ready again to play with his accustomed toys.

Thus the patient with a rapid defervescence of the fever, which in uncom-



plicated cases often lasts a day and a half instead of half a day, reaches the third period of the affection, the period of convalescence. That this must still be looked upon as belonging to the disease and cannot be designated as a return to health, is founded upon the fact that during the period of defervescence symptoms exist which only slowly disappear and that it is particularly this period in which a great number of complications and sequels originate.

The *catarrhal symptoms* are those which now slowly disappear. The conjunctiva still secretes for a little time, the eyelids still suffer from the influence of the secretion, blepharitis ciliaris occurs, slowly the coryza diminishes, but still a number of days must intervene before the voice becomes clear and cough gradually disappears.

The skin shows a tendency to sweating while the exanthem is disappearing and a pigmentation which has remained fades either rapidly or slowly. The skin is still very sensitive to refrigeration, its effect is regularly shown by an implication of the mucous membranes particularly those of the respiratory system.

Besides this, a peculiar late effect of the poison of measles makes itself felt: namely upon the heart. The frequent pulse, corresponding to the rise in temperature, falls simultaneously with the fever and now becomes abnormally slow and irregular. The exact character of this irregularity has not yet been studied; this arrhythmia disappears after a few days without a permanent injury to the heart remaining behind.

The duration of the period of desquamation cannot be given with absolute certainty. There is no absolute indication by which we may determine the return of complete health and normal resistance. Temperature taking is of no avail here for, as has already been mentioned, the period of convalescence lasts for days and in some cases perhaps for weeks longer than the period of defervescence.

In practice rather than to regard this period as too brief, it will be well to prolong it and to consider it as continuing as long as there are still signs of an acute catarrh of the mucous membranes.

From this, the described picture of the regular course of measles, there are a number of complications and deviations, in those concrete cases, the discussion of which alone gives a correct idea of the actual condition of the process in measles.

## DEVIATIONS FROM THE NORMAL COURSE

Even during the *period of incubation*, symptoms may appear, in contrast to the undisturbed health which is usually present. Occasionally this may depend upon accidental conditions, upon catarrhs or other deviations from health which may be due to other causes, such as ordinary colds, influenza, whooping cough, etc. But there are still quite a number of cases in which the infection of measles even during this time makes itself felt by quite a

number of disturbances. This may be recognized from the fact that these symptoms arise during the time at which, according to calculations—for example, the opportunity for infection, the later appearance of the eruption—the infection must have taken place.

These are particularly *slight rises in temperature*, which are noted in children in whom regular temperature observations are undertaken because it is suspected that they have become infected. Without showing any symptoms of disturbance in health, during some days, or in the evening, occasionally for several succeeding days, temperatures of 100.6° F. to 101.1° F. may be noted in the rectum, with this the daily variations are slight and even in the morning the temperature may reach 100.2° F. to 100.4° F.

In other cases, simultaneously with the slight rises in temperature, the children show other symptoms, particularly disturbances of the same kind as those occurring in the prodromal or catarrhal stages of the disease, but of

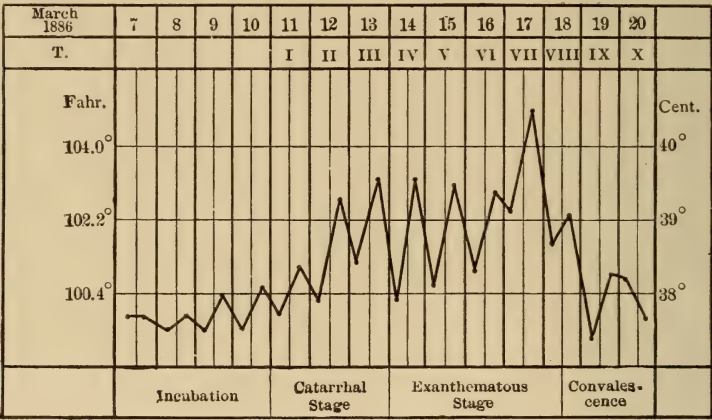


FIG. 22.—TEMPERATURE CHART IN MEASLES.

less intensity, for example, coryza, slight catarrh of the conjunctiva, bloated face, eyes filled with tears or the like. These symptoms disappear and only then, with well developed fever and distinct catarrhal phenomena, does the actual disease begin.

In this manner the disease runs its course, as is shown in the above temperature chart (Fig. 22).

Where there is no opportunity to measure the fever during the period of incubation, we are told that in a case of this kind catarrhal symptoms have been present for a long time. Very frequently the parents state that cough has been present for a number of days previous to the onset of the disease, which began with pain in the eyes and running of the nose.

It may be readily understood that the catarrhal phenomena and, eventually, also, mild fever are subjectively somewhat more pronounced during the period of incubation, but the chronology of the onset of the disease cannot

be determined with certainty from the statements of the patient. Thus, it may be explained that parents say the child has not been well for six, eight or even fourteen days before the appearance of the eruption. Only occasionally does the infection itself appear to be introduced by a febrile period of brief duration which then gives way to an undisturbed period of latency.

An example of this is shown by the following case:

A girl aged seven years, taken ill on October 27th with pains in the throat and lachrymation. Early upon October 24th there was a temperature of 102.2° F., in the evening 103.2° F. Upon October 25th a temperature of 101.1° F.; nothing but a slight redness of the palate could be recognized. Upon October 26th, a temperature of 100.8° F., then complete health; the child returned to school. Upon November 1st, therefore, twelve days later, the prodromes of measles appeared and upon November 4th the exanthem of measles was seen.

In an adult aged twenty-five years, ten days before the appearance of the eruption, I once noted severe *arthritic pains*, beginning in the ankles, then occurring in the knees, shoulders, elbows and hands, later attacking the sacral and lumbar regions, and being accompanied with an irregular, not very high fever. With the appearance of a very intense exanthem of measles all pains suddenly disappeared.

The *invasion*, or so-called *prodromal period*, of measles is often characterized by very slight symptoms. Catarrh of the eyes, nose and trachea are then of a minimal nature, as in a very slight coryza, or they may not even be noted at all. There are cases of this kind where the deviation from the normal can only be recognized when regular temperature observations are taken. Where this is not the case the parents very properly assert that the child was quite well prior to the appearance of the eruption. If, thereafter, the entire affection is very mild and also the fever which occurs during the eruption is of brief duration and slight, even the most experienced may come to the wrong conclusion that he is not dealing with measles at all but with röttheln.

On the other hand, the disease may begin with unusual, severe disturbances in general health. This occurs particularly in very young children, those up to two years of age, then a rapid, high, continued fever appears, occasionally being ushered in by convulsions, intense catarrh, vomiting, diarrhea and coma. This severe condition lasts for several days of the measles process and is often enough followed by severe complications; but these high febrile prodromes are not necessarily of bad prognosis; sometimes after the appearance of an intense eruption defervescence occurs in the ordinary period of time.

Sometimes the initial disease of the mucous membranes appears with extraordinary severity. The conjunctival catarrh assumes the character of a true blenorrhea, the dry swelling of the mucous membrane becomes so marked that the children develop a loud sniffing, dyspneic type of respiration (prognostically an unfavorable sign), or the muco-purulent excretion is so profuse that the children lose their rest from the alternate contamination



and cleansing of the parts. Occasionally very severe epistaxis occurs, being so marked that tampons are necessary. The transference of the disease of the mucous membranes to the Eustachian tube and to the tympanic cavity may even occur in the prodromal stage. The submucosa of the larynx may be so greatly swollen that serious symptoms of pseudo-croup may usher in the disease; the tracheo-bronchitis may give rise to severe difficulties, particularly causing disturbing cough.

Occasionally foreign symptoms develop. Prodromal eruptions appear, with particular frequency resembling those of miliaria. Occasionally the skin, particularly in cases of children with valvular disease, takes on a peculiar marbled appearance before the beginning of the eruption. Sometimes urticaria-like eruptions appear. In children who suffer from slight dry eczema of the face, the affected areas show a peculiar macular measly appearance one or two days before the true eruption begins.

In place of the catarrh of the upper respiratory mucous membranes or simultaneously developed with this, there is sometimes a simple or purulent inflammation with occasional swelling of the lymphatics at the angle of the lower jaw, then the prodromal fever is higher than ordinary.

Finally the catarrhal stage of measles is now and then abnormally prolonged without other symptoms appearing. Thus it may occur that five, six, even seven days pass before the cutaneous eruption becomes pronounced. Often upon the fourth or fifth day it seems as if the eruption would appear, for around the eyes or nose quite sparse, pale non-characteristic macules appear, but they do not become distinct; the eruption tarries until, finally, two or three days later a distinct and marked exanthem arises, which then usually very rapidly implicates the entire body so that the whole process, nevertheless, is not particularly prolonged. Such an extended prodromal stage always shows fever, this is usually of a remittent type, occasionally, however, it may be of a high continued character. So long as the investigation of such cases shows no objective or subjective signs (for example dyspnea) pointing to an internal complication, there need be no fear on account of the tardiness of the cutaneous eruption. Often this condition is followed by very dense confluent or even a hemorrhagic form of the exanthem.

Deviations from the regular course, having a serious prognostic import, are quite rare during the period of invasion or prodromal stage, and for this reason also a separate examination of this stage of the disease is of value. Such complications occur in both of the following periods, that of the eruption and that of defervescence. In these, however, in the main the nature of the serious changes are not equal. In the period of eruption the great catastrophes occur, the breaking down of the resistance of the entire organism; in the period of defervescence the attacks of individual parts which at the onset show but slight damage of separate organs or parts of organs which, however, by distribution over large areas or invading deeply gain an ominous importance.

Regarding the *period of eruption*, deviations in the form of an especially

mild course are to be noted. In a general, mild affection the cutaneous eruption may be slight and of a fleeting character, whereas, simultaneously, all other phenomena disappear rapidly. There are even infections with measles of a benign character without an eruption; this is shown by the following clinical history.

Hessel, Max, aged three years, taken ill, during the night from January 27th to 28th, with fever, lachrymation, conjunctivitis, headache. Upon the evening of January 30th, temperature  $104^{\circ}$  F. January 31st.  $101.3^{\circ}$  F. in the morning,  $104.5^{\circ}$  F. in the evening. February 1st,  $101.5^{\circ}$  F. Nothing could be determined; there was marked swelling and redness of the eyes and coryza, evening temperature  $102.9^{\circ}$  F. February 2d, temperature  $101.7^{\circ}$  F., early in the morning, continued complaints of the eyes, and headache; marked coryza, cough. Evening temperature  $102.6^{\circ}$  F. February 3d,  $101.5^{\circ}$  F., pulse 156. Very restless during the night, much cough, with nausea; evening temperature  $103.6^{\circ}$  F. February 4th,  $100.8^{\circ}$  F., condition the same. Posteriorly, over both lower lobes bronchial râles, also some fine râles, evening temperature  $101.1^{\circ}$  F. February 5th,  $100.9^{\circ}$  F., in the evening  $102.2^{\circ}$  F. February 6th,  $100.2^{\circ}$  F., thence on afebrile course. Complete recovery.

Early upon February 13th, that is, about thirteen days after the onset of the disease, his brother George, aged one year and a half, showed a typical eruption of measles. Max, who had not previously had measles, was not attacked.

In this case, therefore, we are dealing with a febris morbillosa sine morbillis (similar to conditions in other acute exanthemata, such as scarlatina and variola).

The *serious changes* during the *period of eruption* are more important; among these are first to be mentioned those, fortunately rare, cases with a rapidly fatal course. They are analogous to conditions which we meet in scarlatina and other acute infectious diseases and are probably the expression of a most intense intoxication with the virus of measles, or of a lowered resistance of the cells of the body to the poison. Since we have learned, especially by the labors of Ehrlich, Pfeiffer and others, to appreciate the methods by which the organism protects itself in the combat with bacteria and toxins, this explanation can no longer be looked upon as a mere phrase.

In these cases, as, ordinarily, the disease begins with muco-purulent catarrh; in some of my cases high fever was present from the onset, in others the temperature rose gradually. However, from the beginning a marked implication of the sensorium is conspicuous. The children, even during the period of invasion, become decidedly apathetic, sleepy, and lie in bed with their eyes closed, these being often markedly swollen; they have neither appetite nor thirst. Upon the fourth or fifth day of the disease there appear upon the back or other parts of the trunk, some sparse, pale, not very distinct macules, which do not become more developed during the next few days. In spite of this, the fever rises, apathy alternates with nervous unrest; trembling of the extremities, a staring look and strabismus appear; lips and tongue become dry and fuliginous, and now, after a period of from seven to eight days, severe convulsions lasting for hours terminate life, or even without this, death occurs from nervous exhaustion. In the lungs, in some cases, the onset of a bronchitis of the finer tubes may be noted, but this is by no means always





feeble undeveloped form, showing an indistinct bluish color with indistinct demarcated contours, irregularly limited to individual cutaneous areas. During this period the child seems to wither, becomes decrepit, has hollow eyes, cyanotic lips and extremities; only the respiratory function is stimulated and increased. Dilatation of the alæ of the nose, increased respiratory frequency, marked action of the intercostal muscles, besides the movement of the lateral thoracic region, retraction above the region of the insertion of the diaphragm, sometimes general stenotic inspiratory retraction in the jugular and supraclavicular spaces and epigastrium, which almost resemble croup: all this points to a severe implication of the bronchi and the lungs. Examination shows the signs of a gradually extending capillary bronchitis; the heart becomes weaker and weaker, and often with the addition of convulsions, sometimes also of profuse diarrhea, whereas the entire skin becomes flaccid and from the eighth to the tenth day of the disease the fatal issue results. The autopsy then shows the cause of the respiratory affection to be a disseminated inflammation of the mucous membrane of the entire bronchial tree, contrasting markedly with the pallor of the skin. A large number of the finest bronchi are plugged with mucus and the onset of the inflammatory and atelectatic changes in the lungs can always be determined. The entire process in fact gives a complete impression that the poison of measles, which under normal circumstances produces inflammatory changes in the skin, has taken a false passage and has implanted itself upon the mucous membrane of the bronchial tree. From this first form of the fulminant fatal course, a second one is differentiated in that here a proper resort to treatment may favor the regular localization of the measles poison, whereas in the first instance all therapy is unavailing.

To these general catastrophes I should like to add a third which particularly implicates the lungs, but which in the main is but little recognized, and which, in my opinion, must also be referred to the severity of the measles virus. These cases are rare but I have met with them in several separated measles epidemics. They may be designated as a *rapid necrotic pulmonary inflammation due to measles*. The following clinical history will exemplify the course of this form.

Bode, Emilie, aged one year, both parents healthy; the child having been completely well up to then was attacked, on April 19, 1877, with the usual symptoms of measles, which simultaneously attacked another child living upon the same floor of the house and which in the latter child ran a regular course. With conjunctivitis, which rapidly took on a quite intense character, coryza and cough were present, but no distinct eruption appeared. Moreover, with the fever which was at times high and moderately remittent, on April 25th, over the right, and on April 27th also over the left lower lung, signs of a quite diffuse infiltration appeared which, with continuing fever, caused a marked decline of strength, with frequent collapse, and then attacked the upper lobe. The child became more and more apathetic, complete anorexia occurring, and with closed eyes moaned and complained. Frequent spasmodic grinding of the teeth. Quite severe general condition. Thus the affection lasted several weeks, until May 14th, twenty-six days after the onset of the disease, an eruption of measles appeared, which, however, upon the following day became pale and cyanotic. With increasing asthenia and cardiac dyspnea the child died upon the same day, May 15th.

The necropsy showed the following: The right upper lobe of the lung was adherent to nearly the entire thoracic wall by firm pleuritic adhesions. As was shown by section, it was transformed into masses of cavities containing pus, of the size of a large hazelnut, which proved to be dilated bronchi with very thin walls. The pulmonary tissue lying between, as well as that of the middle lobe, was changed into a mellow whitish-gray to yellow, discolored tissue (but not caseous), in which the original structure could still be discerned, but which showed a decided reduction from the original volume. The left upper lobe was but little altered. Both lower lobes showed the usual condition of disseminated lobular pneumonia of a yellowish-brown color. In these lobes also a high-graded purulent bronchitis could be noted. The bronchial glands were markedly swollen, medullated, bluish-red; nowhere caseation nor miliary tubercles. Spleen decidedly swollen, pulpy, dark bluish-red. Both kidneys considerably enlarged, with marked yellowish discoloration of the cortical substance; slight intestinal catarrh.

We see, therefore, a child previously healthy, taken ill with prodromal symptoms of measles, and instead of the eruption, a pulmonary affection of a quite peculiar character appears, which in four weeks leads to death; shortly before this a fleeting measly exanthem makes its appearance. Two processes in the severely affected lungs here act together: first, the acute necrosis of the tissue. (In cases occurring later I made a histological examination of the pulmonary tissue and found a non-nucleated appearance of the alveolar exudate as well as of the original dense cellular infiltrated pulmonary tissue.) Secondly, a quite rapid development of bronchiectasis not so much due to an ulceration or suppuration as to a thinning and loss of permeability of the bronchial wall. Taken altogether, it is a quite acute and severe lesion which implicates the pulmonary and bronchial tissues to a pronounced extent. It was of great interest to me some time later (at a meeting of the Society of Naturalists in Hamburg) to see some specimens of pulmonary plague, in which there was the same combination of acute bronchiectasis and necrotic decomposition of the inflamed pulmonary tissue.

In the epidemic of the year 1877 I saw two cases quite analogous to the one described, but with a still more rapid course, they were, however, not so pure as the ones described above, as, besides, caseous bronchial glands were found. In spite of this the acute pulmonary necrosis was not due to tuberculosis. Later I met with similar cases from time to time. I should like to bring these into analogy with the acute inflammatory tissue necrosis of the palate in scarlatina, that refers this development directly to the poison of measles, for the ominous change, as already mentioned, occurs particularly in the beginning and at the height of the disease, at which time mixed infections are not so frequent. Naturally, by this it is not meant to deny that the latter also, under some circumstances, may lead to the severest injury of tissue. I observed a case of necrotic pneumonia, besides malignant endocarditis with cutaneous hemorrhages and renal infarcts, in a nursling aged ten months. The entire process, however, in this case only developed fourteen days after the eruptive period.

This pneumonic form of measles certainly deserves further study; it is sufficient here to call attention to the course, which up till now has been little observed. The prognosis is always ominous to the highest degree.

Before leaving the description of the eruptive period of the disease, *another variation of the exanthem* must be briefly considered. The form which is of little prognostic importance, as well as the important rudimentary forms, the smooth and elevated forms, and the hemorrhagic consistence of the eruption, these have already been described.

The formation of nodules in the individual macule occasionally reaches such an extent that the appearance of the eruption is entirely altered. In each macule that is at all round, a nodule occurs, but of such a size that the face, for example, becomes covered with dense, cloudy, markedly red prominences. The appearance of the patient will then show great similarity to variola. In the case of a young child, upon its buttocks which have become irritated by feces and urine, the nodules in the measles macules spread out, form lentil-sized flat infiltrations, with umbilicated surfaces, and, after the epidermis has become desquamated, are of a shining flat consistency, which may cause confusion with syphilitic eruptions.

Sometimes the exanthem, by a special variety of confluence, shows large macules, the individual spots reaching the size of a ten-cent piece, even the size of a quarter. Sometimes irritation of the entire macule occurs, which then stands out above the other skin like a high plateau, the whole condition very markedly simulating urticaria.

Occasionally in these large individual nodules of measles I have noted pustule formation, which may cause still further errors in diagnosis. The miliaria form of the measles exanthem has already been mentioned.

In some cases, on the other hand, the macules permanently remain as small spots, without confluence, the color then remains light red and the entire appearance of the eruption may bear great similarity to a feebly developed scarlatina.

The relation of measles to pemphigoid cutaneous eruptions is very remarkable, and has been lately emphasized by some authors. I am able to confirm these reports. Not only that not infrequently an eruption of vesicles resembling pemphigus appears in connection with the eruption of measles, but that the eruption of measles itself may appear in the form of a pemphigoid exanthem and run its course.

This is proven by the following case:

On October 23, 1881, George Schilling, aged three years, was attacked by the prodromes of measles, the normal exanthem following, and upon October 26th his brother Herman, aged seven years, was attacked in the same manner.

Simultaneously upon October 26th, their sister Louisa, aged nine months, was attacked with unrest, fever, coryza, hoarseness. Temperature upon October 27th, 100.6° F., in the evening 101.5° F. October 28th, 102.4° F. Upon the face, upon the neck and upon the upper trunk a large number of warty sore spots, partly flaccid vesicles, filled with a thin fluid, being the size of a pea and larger and resembling pemphigus. October 29th, temperature 104.9° F.; the vesicles have not increased, evening temperature 103.2° F. No measles eruption, only upon the buttocks some elevated redness. Evening temperature 105.1° F. October 30th, morning temperature 103.2° F., evening temperature 102.4° F. October 31st, temperature 101.8° F., evening temperature 99.5° F. November 1st, 100.4° F. The vesicles have increased and enlarged, upon the back



some the size of a walnut are noted. Upon the buttocks and posterior surface of the legs a raised erythema. Evening temperature 100.6° F. November 2d, 100.6° F., upon the trunk a few new vesicles. Now also upon the lower thighs a few vesicles, evening temperature 99.9° F. November 3d, temperature 99.1° F., a few new vesicles upon the trunk. The old had for the most part ruptured and given rise to erosions. November 4th, no fever, formation of vesicles had ceased, hoarseness still existed.

Of interest, further, is the occurrence of a *relapsing eruption* in measles. I saw this in a girl aged four years, who in the course of a month had two eruptions, and a month and a half later, for the third time, showed an exquisite eruption of measles.

Upon April 16th she was attacked by cough, upon April 27th conjunctivitis with slight fever occurred, and then upon May 1st, with but a single rise in temperature, to 102.7° F., an eruption appeared which distributed itself over the entire body.

Upon May 30th, after all traces of the eruption had disappeared, a second exanthem occurred which was taken to be rubella.

Upon July 15th, the child was taken ill for a third time, with headache and coryza, and, again, upon July 19th, a quite characteristic measles eruption occurred which lasted ten days.

What appears remarkable was that each of the attacks was conspicuously mild regarding fever and accompanying phenomena. Other authors have also described these relapsing forms.

The *desquamation of the skin* after the disappearance of the eruption is very slight and is often entirely concealed by the return of sweating during the stage of convalescence. Fine flaky desquamation of the epidermis may be noted upon the face and neck, upon the buttocks, and not infrequently upon the lower portions of the legs.

Occasionally very marked peeling occurs, particularly in the face, which will then last for days, and may even assume a large lamellar character upon the temples and forehead.

We now reach the deviations in the course and in the *period of convalescence*. This is the true domain of the manifold local disturbances which the entire pathologic process suffers and which, often beginning insignificantly, in their further development reach a life-threatening severity, at other times, naturally, also, beginning abruptly in the course of a few days.

Even where we do not discover the point at once at which the regular process of the reparative changes has been interrupted, there is always a symptom which may be looked upon as a signal of alarm and which under all circumstances should cause a careful investigation: This is an insufficient defervescence or the renewed rise of temperature in the period of convalescence. It cannot be denied that occasionally a certain hesitating drop of the fever by lysis, or also a mild post-febrile course may occur without it being possible to determine a complication, but this is certainly not frequent and, as a rule, the previously mentioned variation of the body temperature may be looked upon as a sign of irregularity in the course of the disease which may cause anxiety for days and weeks.

To present a well-ordered summing up regarding the manifold changes that may occur, it is necessary to present them in a topographico-anatomical review in which it is necessary to remember that often in the individual case quite a number of these secondary conditions may combine.

*In the eyes*, the catarrh of the conjunctiva is often prolonged. Particularly in somewhat "scrofulous" children the mucous membrane inflammation which leads to the gradual agglutination of the lids may last for weeks and even for months; a stubborn blepharitis ciliaris is added, which, again, is followed by eczema in the surrounding tissues of the eye. Gradually there develop, in case treatment is not effectual, corneal phlyctenula and superficial ulceration, with photophobia, blepharospasm, in fact, the entire symptom-complex of "scrofulous" ophthalmia.

Sometimes, however, the catarrh runs its course as a severe purulent conjunctivitis and marked painful edematous swelling of the lids, a true blennorrhoea of the mucous membrane of the eyes. I once saw the sad case of a boy who in the short period of a two-weeks attack of measles lost the sight of both eyes by a purulent pan-ophthalmia. The desperate father wanted to sue the physician who treated the child, before it was admitted to the hospital in Leipzig, for he believed—but quite unjustly—that neglect was the cause of the inflammation of the eyes.

More manifold and numerous are the affections of the nasal mucous membranes. An infrequent but unpleasant, and, according to my experience, unfavorable, prognostic course, is shown by the ordinary coryza of measles, when dry swelling of the mucous membrane which may be referred to a more marked inflammation of the submucosa takes place. The children snuffle—it always occurs in earliest childhood—and when drinking or even otherwise when closing the mouth this becomes loud and prominent, the alæ of the nose retract a little with every deep inspiration. Examination shows that there is no secretion in the nose, the opening is covered with dry brown crusts and partly closed.

At other times there is a profuse purulent secretion from the nose, which often predominantly affects the nasal cavity, then this and its surroundings become excoriated, deep ulceration occurs, the adjacent skin often swells to a great extent and shows a hard and glistening upper surface. A smeary or lardaceous exudate is deposited upon the ulcers, ecthyma-like eruptions appear in the vicinity, which again ulcerate or are covered with thick crusts; a process which is accompanied by continued fever, markedly exhausting the affected individual, and while not in itself leading to a fatal outcome, nevertheless causes prolonged invalidism. This condition may be designated as *diphtheroid* of the nasal mucous membranes and its surroundings. Frequently also the surroundings of the mouth take part in this ulcerative process and often complications, profuse diarrhea or pulmonary involvements, are added and may then lead to a lethal outcome.

The distribution of the infectious mucous membrane affection from the nasal cavities of the posterior pharyngeal wall, and, above all, to the Eu-

stachian tube, is still more important. If the inflammatory process has once reached these tissues it rapidly distributes itself to the tympanic cavity and we then have the so very frequent complication of *measles with otitis media* to consider.

This shows itself, almost always, by a new rise of the fever, often to a marked height, 104° F., and over, even above 105.8° F., the character, however, being remittent, so that usually in the morning remission occurs, the temperature falling to 102.2° F. or lower, even reaching the normal. The general condition again becomes worse, loss of appetite reappears and to this a certain unrest is added which is especially increased at night. Older children now usually complain of stitches or other pains in the ears, but by no means always, frequently there is a complaint of headache accompanied by hebetude and delirium at night. Small children usually moan continuously, but this is not always pronounced. Not rarely is the peculiar oblique position of the head seen in a unilateral otitis media, especially where, in connection with this, a painful lymph gland swelling develops under the sternocleidomastoid of the same side. The ear speculum rapidly shows the cause of these symptoms. The tympanic membrane has lost its normal lustre, the light reflex has disappeared and, mostly, in the surroundings of the handle of the malleolus, red or grayish-red swellings are noted. Not rarely may a yellowish or yellowish-brown exudate be seen shining through the tympanic membrane. The pus which usually collects rapidly in the narrow space of the drum after a few days usually ruptures externally through the tympanic membrane. The point of perforation is most frequently situated in the posterior lower quadrant.

Where this spontaneous rupture does not occur, the catarrh of the tympanic cavity with a moderately severe, more mucoid, inflammation may leave spontaneously, the fever disappearing by lysis. If, however, the fever reaches to its former height, then the artificial opening of the tympanic membrane must not be neglected, for, otherwise, further dangers threaten. The infectious inflammation attacks the antrum, insidiously implicates the cells of the mastoid process, and from here easily reaches the transverse sinus or the dura mater or pia mater. I have several times seen a septic sinus thrombosis or subsequent pyemia due to an otitis media, which, a few weeks after the disappearance of the exanthem, terminated the life of the patient.

Or the affection becomes a chronic otorrhea with carious degeneration of the bone, which, by causing suppuration in the brain much later, may result in death. This serious change in the otitis of measles may be prevented by timely interference. Painfulness, slight swelling, or redness of the mastoid process should always receive attention, and, even when these signs are absent, sometimes alone upon the basis of a fever that cannot be otherwise explained, a marked protrusion of the posterior wall of the external auditory meatus, or a very profuse suppuration, we must come to the conclusion to have the mastoid process of the temporal bone opened.

The *oral cavity* may also be implicated in measles. Not very frequently



there develops, especially, beside other complications, stomatitis of the mucous membrane of the palate, gums or cheeks. Mistakes may be caused if the mycosis appears in those areas in the period of convalescence of measles, in which in the prodromal stage the characteristic spots have been noted. This eruption is of little importance and is readily removed.

*Aphthous stomatitis* occasionally gives rise to great difficulties. These painful eruptions may occur in all parts of the mucous membrane of the mouth, they occur particularly upon the gums of the incisor teeth and upon the adjacent inner surface of the lips, as well as upon the anterior part of the tongue, and often result in a very marked edematous swelling of the lips with formation of hemorrhagic fissures, also similar ulcers in their surroundings, as has been described under nasal diphtheroid.

At other times more circumscribed but ulcerative inflammations occur in individual areas of the mouth and from them hemorrhagic infiltrations of the mucous membrane and mucosa are distributed along the gums. The severest form of this secondary mouth affection leads to noma which has a particularly close relation to measles. The description of this condition will follow later on.

Finally, *the larynx* must be considered. That this organ in the first days of the disease may also give rise to symptoms of pseudo-croup has been mentioned; much more frequently, however, in the stage of convalescence there is a disturbing increase of the laryngeal catarrh. This usually occurs in young children from one to two years of age.

Under continued and, occasionally, very high fever (over 104° F.) the hoarseness which was already present during the stage of eruption changes into complete aphonia so that even crying occurs quite without tone. Beside aphthous oral affections, also ulcerative nasal and lip affections of the variety described above may increase the difficulty. The children always become restless, do not sleep, throw themselves about, and it usually requires a few days until the stenosis of the larynx reaches a certain grade. Respiratory retractions which are not very decided but are still worthy of note occur in the jugular space and in the epigastrium, especially if the child is irritated and as a consequence of this breathes rapidly. Then the inspirations and expirations are accompanied by those sounds which denote a beginning narrowing of the opening of the glottis. Inspection of the oral cavity shows reddening and soon also marked or slight swelling of the palatine and pharyngeal parts, but—in case aphthæ are not present—no deposits or membrane formations can be noted. Now anxious hours and days begin. Prior to each visit the physician believes that the time for intubation or tracheotomy will have arrived, and yet no increase of the phenomena of stenosis can be determined. External palpation of the larynx and of the trachea teaches that these parts are very sensitive to touch—therefore, by the superficial plugging of the cartilage, perichondrium and connective tissue they are in a condition of inflammatory infiltration. This clinical picture just described in fact depends upon an inflammation of the mucous membrane of the larynx and the surrounding parts (particularly

the *submucosa*), which is not infrequently confirmed by the autopsy. The changes which are found in the cadaver in cases of this kind are, a rigid, thickened epiglottis, prominent false vocal cords and arytenoid cartilage coverings, all showing a deep dark redness and being permeated by hemorrhages and showing a velvet-like swelling of the markedly reddened mucous membrane of the trachea from above downward to the bifurcation. It is, therefore, the severe *inflammatory* swelling of the supra- and subglottic covering of the membrane which causes this form of the disease that, clinically, may well be designated as laryngeal croup. It is also well in the treatment of cases of this kind to adhere to this conception for they are effectually influenced by a proper antiphlogistic therapy (blood letting).

Croup occurring in measles, with *membrane formation* upon the vocal cords and upon the rest of the laryngeal mucous membrane, according to my observation, owes its origin to a *diphtheritic* infection, and for this reason will be considered later on.

In older children the laryngeal affection does not readily assume such a threatening character as in the very young, for, in the first place, the phlegmonous inflammation does not readily lead to stenotic phenomena on account of the greater space of the glottis, and, secondly, the danger of the addition of distributed inflammatory foci in the lungs is a slighter one. Here, occasionally, the very marked tenacity of the hoarseness, which may lead to complete aphonia, and of the laryngeal cough, which may last for weeks and even for months, gives rise to fear. But, finally—at least in the cases that I have seen—complete restitution occurs. Anatomically the condition is due to a simple chronic catarrh without ulcer formation.

It is now generally assumed, and I favor this view, that all of the enumerated complications and distributions of the catarrh, which were originally due to the toxin of measles, are no longer results of this but are due to the addition of new deleterious effects, and especially to the action of secondary bacterial infections. It does not appear that we are dealing so much with peculiar forms as with those generators of suppuration and inflammation which are found everywhere and particularly in the nasal and oral cavities of both the healthy and the sick: staphylococci, streptococci, pneumococci, coli varieties and the like. The possibility of their successful onslaught is prepared by the attack of measles and perhaps particularly increased by external physical deleterious influences (refrigeration).

The same is true also of the numerous and especially important affections on the part of the *bronchi and lungs*, which appear unexpectedly in the period of convalescence. Often, however, these disturbances have already begun during the acme of the eruption and increase later; nevertheless, it frequently happens that nothing points to the complication which develops later on.

The most dangerous form under which the pulmonary complication appears is the catarrhal inflammation of the smallest bronchi, which rapidly distributes itself over the entire bronchial tree, *acute capillary bronchitis* (bronchi-

olitis, suffocative catarrh). It attacks particularly weak children in the first and second years of life, but it by no means spares older children. Thus I once saw a boy aged seven years, for over a week after measles had disappeared show a dyspnea with 80 respirations per minute, being in an exceedingly dangerous condition.

The prodromes are usually without alarming phenomena, often being accompanied with but slight symptoms; the eruption has usually become pale but has appeared over the entire body and then shows a confluent character over the trunk—then the general condition of the child changes. He becomes conspicuously pale, while the remains of the exanthem take on a livid color, the child being restless and very dyspneic; the pale semitransparent alæ of the nose move regularly, the diaphragm heaves, drawing the lower portions of the thorax inward, and the auxiliary muscles of respiration enter into action. The fever does not run very high, hovering about 102.2° F., the pulse, however, shows an unproportional high frequency, rising to 160, 180, even 200 per minute. An examination of the lungs at the onset shows but little regarding the severity of these serious symptoms of disease, but from day to day numerous fine râles are heard, first in the lower posterior parts of the lungs, soon rising higher and higher, and then also being heard in the lateral aspect of the thorax. Tracheal râles are heard at a distance, the cough which becomes feebler and feebler is no longer sufficient to bring up the contents of the bronchial tubes. The unrest and anxiety give way to semiunconsciousness, and the patient lies with half-closed eyes, the bulbæ being turned upward; occasionally general convulsions occur, the pulse becomes smaller and smaller, and gradually life ceases after but a few days of this condition. At the autopsy no changes, or but slight ones, are found in the lungs, but the smaller and finest bronchi are filled by a tough muco-purulent secretion from the inflamed mucous membranes. Two-thirds to three-quarters of the bronchial tree may show this condition; the anterior upper parts are most resistant and remain permeable to air.

The course is less rapid if the inflammatory mucous membrane affection does not disseminate itself rapidly over the greatest portion of the bronchi but limits itself to one or the other pulmonary lobes. Then, primarily, a *circumscribed capillary bronchitis* arises, which, with a somewhat prolonged duration, is always connected with the sequelæ of catarrhal pneumonia in one area, atelectasis in a second, and alveolar dilatation of a third point. At the necropsy a high grade hyperemia of the vessels in the pulmonary area may be determined, from which it is concluded that during life a large part of the amount of blood has accumulated here and has become unsuitable for the function of internal respiration, besides having caused increased labor in the mechanism of the circulation. Finally, the rapidly appearing cellular infiltration of the pulmonary tissue in the surroundings of the diseased bronchi reaches such an extent that the inflammatory irritation has not only affected the surface of the mucous membrane but has permeated into the depths of the same.



The clinical picture is somewhat less fulminant than in the former instance; it is true, fever, increased rapidity of the pulse and dyspnea show the nature and the seat of the affection which has followed measles, but the fever, although it may show exacerbations, is markedly remittent and even intermittent, and the other symptoms are of a more moderate grade. Physical examination usually reveals the principal pulmonary portion affected; profuse loud, coarse, and fine râles are noted usually in the dependent portions of the lower lobes, to which there is rapidly added a diminution of the resonance on percussion, with the appearance of broncho-vesicular breathing or even pure bronchial breathing, which completes the diagnosis of the implication of a more or less large pulmonary division. The general condition is commonly greatly disturbed, lassitude, irritability, anorexia and insomnia being accompaniments of the local condition. A distressing cough helps to diminish the powers of the little patient, and the condition is made worse by inflammations of the mouth, severe laryngitis or by intense intestinal catarrh. However, this form of morbilli pneumonia after a series of several days often enough terminates in recovery.

The favorable outcome is less frequent in those cases in which the severity of the infection rapidly shows a distribution of the inflammation over an entire lobe of the lung or frequently over two or more lobes. This may usually be recognized in a very brief time as the physical signs develop very rapidly in this condition. In such instances, similar to lobar fibrinous pneumonia, there is marked dulness upon one or both sides which may also take in the lateral aspect of the thorax, sometimes also showing lobar catarrhal infiltrations of an upper lobe; with this there is marked bronchial respiration in the area of dulness, besides which, however, many râles may be heard at various areas. The fever has a more continued character and is high, but it may also assume another form. The pulse is very frequent, the respiration sighing and it appears to be painful, also upon palpation and in percussion of the affected parts the patients complain of pain. Exhaustion occurs more rapidly than in the previously described variety; the tongue becomes dry. The lips show sordes; frequently galloping respiration accompanied by sighing; in older children there is prolonged delirium, in younger children convulsions occur. Anorexia is complete, but severe thirst compels the children to consume the fluid nourishment which is given them. The urine is scant and concentrated. In spite of the severity of these phenomena, even this lobar form of post-measles pneumonia is endured longer than the first described capillary bronchitis; strong children may even finally recover from the condition. It is worthy of note that after defervescence the absorption of the exudate in a pulmonary lobe which has been infiltrated in this manner may require weeks and even months before complete recovery finally takes place.

But, unfortunately, as may be only too frequently demonstrated by autopsy, it is seen that in fact the condition is due to a lobar pneumonia, of two or even three lobes, in which, however, the original nature is shown by

the lobular appearance of the section; besides, the character of the exudation is not infrequently of a mixed nature: fibrinous, hemorrhagico-fibrinous and catarrhal exudations are found side by side and intermingled. The pleura in these cases is always found implicated; thin, fibrino-purulent deposits, and inflammatory cloudiness are found in wide areas; beneath the deposit the pleural tissue is permeated by numerous round hemorrhages from the size of a pin-head to that of a lentil.

Exceptionally large, purulent, or sero-fibrinous, or even hemorrhagic exudates develop.

*The digestive organs* do not remain free from secondary infection, although this is much rarer. It has already been mentioned that in all intense infections in measles the mucous membrane of the small and large intestines, and especially their lymphatic apparatus, are always implicated. Besides the previously described inflammations of the mouth, the larynx, the bronchi and the lungs, there are invariably found in the cadaver more or less well-developed signs of an implication of the intestinal tract.

Occasionally the digestive tract is attacked alone, whereas the respiratory organs are spared. In some cases, not alone in nurslings but in older children, the entire affection begins, in addition to the usual catarrhal phenomena, or even instead of these, with severe vomiting and watery discharges which occur frequently, five or six times daily. In the further course of the affection vomiting ceases but the diarrhea continues during the entire course of the disease and only desists with the disappearance of the exanthem. This diarrhea does not appear to have a particularly unfavorable influence upon the course of the affection. In the case of a child aged two years a severe pneumonia was added, and while from the paralyzed anus the thin yellowish-white contents of the intestinal tract flowed continuously, the child succumbed to the pulmonary inflammation.

More frequently diarrhea occurs during the time of critical defervescence, which may almost be looked upon as a critical discharge. Without markedly influencing the general condition during the time of the disappearance of the exanthem, there are muco-hemorrhagic or muco-purulent stools, being small in amount but occurring in rapid succession. The condition, however, is of brief duration, a day to a day and a half, then it disappears, often without treatment. At other times, during the period of convalescence, a few days after the disappearance of the eruption, with the addition of fever, severe abdominal pains, and general illness, a marked diarrhea sets in, with thin, foaming, profuse, at other times sparse, frequent mucous discharges. With suitable diet and treatment this condition is controlled, but usually somewhat prolongs convalescence.

But this is not always the case. Instances occur in which the intestinal affection shows great severity, there is marked tympanites and painfulness of the abdomen; with this there is high fever, severe implication of the sensorium, a dry tongue, briefly, a condition which markedly resembles the typhoid

state. In a case of this kind, in my clinic, I found at the autopsy a severe enteritis with septic processes.

This diarrhea is always unpleasant and of questionable prognosis if it occurs in connection with any severe complication, especially if it implicate the lungs. It requires particular attention, and in young children readily leads to a life-threatening exhaustion provided we do not succeed in rapidly removing the condition.

Finally, the intestinal affection may assume the character of a true dysentery, in a clinical as well as in a pathologico-anatomical respect. It is, however, not unlikely that affections of this kind are not due to the usual but to the specific pathogenic agent. In the year 1892, a child simultaneously suffering from measles and a severe dysentery, was admitted from a children's asylum to the Leipzig Hospital, to the division for measles. In a brief period this affection was transmitted to six other patients, and of the seven children attacked in this manner, five died. The autopsy showed the same changes, a severe hemorrhagic inflammation with a distributed coagulation necrosis of the entire mucous membrane of the large intestine (diphtheria in an anatomical sense), that I saw during the Franco-Prussian War in soldiers suffering from dysentery during the sieges of Metz and Paris.

Threatening symptoms rarely occur on the part of the *nervous system*. A moderate grade of implication of the sensorium and of delirium during the mildly febrile exanthematic stage belong to the clinical picture of measles. In nervous children these deliria sometimes occur upon the first day of the initial fever and accompany the period of eruption even if the temperature does not rise very high. In a child aged three years I saw a delirium appear with an evening temperature of 102.7° F.

Severe stupor, a soporous condition, or wild delirium are found more frequently in adults than in children during the acme of the eruption.

Rarely these severe toxic symptoms also occur in children.

In quite a fat girl aged seven years, who had a very intense eruption, I noted two days of complete confusion with severe delirium following the day of the acme of the disease. The girl rose in bed, endeavored to run from the room, cried and fought, had delusions of fear with slight hallucinations which resembled alcoholic delirium, saw a number of maggots moving about upon the ceiling and the like. Gradually she became quieter and, finally, after a brief period she recovered completely.

The delirium occurring during convalescence is more remarkable, resembling the post-pneumonic disturbances of adults.

In a boy aged six years, at the end of the eruptive stage, in the morning after a fall of temperature (from 104.4° F. to 99.9° F.), there occurred a delirium with hallucinations. He thought that he heard drums and ran to the window to see the soldiers, then from all four corners of the room storks appeared which bit him in the leg; he began to rave and curse, and pulled the hair of his father, who was holding him. This condition lasted the entire day up to five o'clock in the evening. After the use of



chloral quiet appeared. During the next days signs of hallucination with mild fever recurred and then the complete normal condition returned.

A more serious symptom is the occurrence of *convulsions* in the course of measles. In the stage of invasion they are rare and, according to Trousseau's experience, are not of unfavorable prognosis. Where, on the other hand, they occur at the time that the eruption should appear, which almost exclusively happens in children from one to two years of age, they are very serious and indicate a very severe general intoxication which almost invariably results in death. I have occasionally seen this in the stage following the eruption, in the second week of the entire affection, as an accompanying symptom of pulmonary or bronchial affection—but not with the same unfavorable prognosis. Often the cases terminated in recovery.

It will always be well to give a guarded prognosis even in these post-morbilli convulsions, in view of the experience that measles belongs to those infectious diseases in which encephalitic conditions may arise, for, prior to leading to paralyses, they show themselves first in the form of convulsions.

*The skin* is not infrequently implicated in the measles process, apart from the described variations of the eruption. Once, in a child aged two years, I saw convalescence retarded by a very intense eruption of miliaria. Occasionally, in children who have been weakened by diarrhea or chronic pneumonic affection, stubborn furunculosis arises, also eruptions resembling pemphigus may appear even a long time after the disappearance of the measles exanthem.

In septic endocarditis, hemorrhagico-necrotic foci of a circumference of several centimetres appear upon the abdominal wall or also upon other parts of the surface of the body.

The severest cutaneous affection, upon the whole very rarely occurring in a large number of cases in individual epidemics, is *noma*, *cancrum oris*. Its point of selection is the skin of the cheek and the surroundings of the female genitalia, the large labia and neighboring parts. I once saw it arise from the floor of the mouth beneath the tongue, in a tuberculous child suffering from measles and whooping cough. The point of entrance for the pathogenic agent of this gangrenous condition, which advances with dreadful rapidity, always appears to be an ulcerated area in the mucous membrane, either in that of the cheek near the angle of the mouth, or in the vulva. The poison rapidly passes through the tissues; soon an elastic edema is noted in the affected area of the face opposite the mucous membrane of the cheek, in which first there is a brownish then a blackish area, indicating complete necrosis of the affected portion of the cheek. While this decomposes to a slimy gray green, dreadfully offensive mass, the gangrene advances into the depths of the tissues and into the surrounding areas, usually in a manner quite painless to the child, but with increasing, finally fatal, exhaustion. For the most part the affection occurs in children that are otherwise cachectic. Regarding the claim lately made connecting the affection with the pathogenic agent of diphtheria, further investigations are necessary.

Occasionally *rheumatic*, *arthritic* and *cardiac affections* occur in connection with measles, but much more rarely than in the case of scarlatina.

In a somewhat debilitated boy aged eight years, five days after the appearance of the eruption, I saw renewed fever, and two days later both hip joints were attacked with extraordinarily severe pains. Even lifting the bedclothes led to a scene of crying, and an accurate examination of the markedly retracted legs was impossible. We considered it a severe purulent arthritic inflammation. Simultaneously, a soft systolic murmur could be heard in the heart; in the course of four days both anomalies disappeared completely under the use of sodium salicylate.

However, even without inflammation of the joints, in the period of convalescence, benign endocarditis occurs, from which recovery takes place, but under some circumstances it may even lead to permanent valvular disease. Malignant endocarditis is found in individual cases after an especially severe case of necrotic measles pneumonia.

*The kidney* is but very rarely damaged; at the height of the severe febrile exanthematic stage toxic albuminuria is noted, which, however, rapidly disappears; in the post-eruptive stage the kidney is spared.

In those rare cases in which an infectious nephritis accompanies measles the conditions are the same as in hemorrhagic desquamative scarlatinal nephritis, and, similarly, may remain as a chronic affection.

## COMBINATIONS OF MEASLES WITH OTHER SPECIFIC ACUTE OR CHRONIC INFECTIOUS DISEASES

Measles may be combined with other *acute exanthemata*, in that they may either precede or follow the condition, or also that they may run their course *simultaneously* in the same person. I have seen the latter condition in the case of varicella, vaccinia and scarlatina. In such cases we have the impression that both infections run their course in the same organism without particularly influencing one another. At most, one or the other may show a briefer course than usual. For example:

A boy aged six years was taken ill upon November 20th, with cough and coryza. Remained in bed. Upon November 23d, complained of headache, difficulty in deglutition and hoarseness. Evening temperature upon November 25th, 102° F.; upon November 26th, in the morning 101.5° F., evening 103.4° F.; pains in the eyes. Right tonsil swollen and reddened, pharyngeal catarrh. November 27th, eruption of varicella; morning temperature 101.8° F., evening temperature 103.4° F. November 28th, varicella eruption disappearing. First eruption of measles in the face; temperature 102.9° F., in the evening 104.2° F. November 29th, maximum temperature 103.6° F., evening temperature 103.4° F. November 30th, temperature 103.1° F., evening 100.8° F. December 1st, temperature 99.1° F., eruption of measles disappeared.

Similar conditions may occur in vaccinia.

A girl aged four, who for two years had suffered from infantile cerebral paralysis, upon May 27th was vaccinated while she was in the stage of incubation of measles.

June 3d, coryza and cough. June 5th, eruption of measles appeared and developed to its maximum to June 7th; temperature 104.7° F., evening 104.4° F.; June 8th, 102.9° F., evening 100.9° F. The vaccine pustules began to dry. June 9th, 103.4° F., evening 101.1° F., eruption becoming pale. June 10th, 100.8° F., evening 100.2° F. June 11th, 101.8° F., evening 99.9° F. Afebrile thence on. No reason could be found for the "after fever" of measles. It might be referred to vaccinia.

At other times measles disturbs the course of vaccinia, provided the affection occurs soon after vaccination, the measles process itself not remaining uninfluenced, as, for example, in the following case:

Boy aged fifteen months, vaccinated upon July 2d. Upon July 3d, conjunctivitis and cough. The eruption of vaccinia does not develop properly. Only upon July 9th does the eruption of measles show itself, to disappear upon July 14th; during the entire eruptive period, fever (up to 103.5° F.) and marked dyspnea are present.

Upon July 10th, the eruption of vaccinia appears as it would in other cases upon the fourth or fifth day; upon July 12th, the areola appears, the pustules being well developed upon July 14th. On July 15th desiccation occurs. Continued fever which is prolonged to July 24th by a suppurative otitis media.

To diagnosticate the simultaneous course of measles and scarlatina is always very difficult, still I believe I have observed cases of this kind, the double character of which could be discerned by the symptoms and the subsequent sequels. I have certainly observed the appearance of both exanthems at the same time in the same family. A girl aged one year showed a normal attack of measles (catarrhal symptoms being present during the stage of incubation) without being affected by scarlatina before or afterward, whereas two older children suffered from scarlatina.

If the exanthems succeed each other, the course of the affection depends in the main upon whether the severer infection or the milder one follows. In the former case the prognosis is always uncertain. Measles following varicella more readily takes an unfavorable course and shows complications than vice versa. Naturally, if varicella occurs during an irregular attack of measles this is capable of aggravating and of prolonging an existing bronchitis or pneumonia.

Of 10 cases of measles in my clinic, occurring in connection with scarlatina, 1 died. Of 10 cases of scarlatina following measles, 4 died.

Measles may combine with other acute cutaneous eruptions. Twice in the same epidemic I saw a very intense erythema exsudativum multiforme appear in the stage of convalescence of measles; once eight days after, the second time twenty days after the maximum of measles had been reached, in both cases lasting several weeks. Herpes zoster, etc., also occurred.

According to my experience, one of the most serious combinations is that of measles with *diphtheria*. I cannot escape the impression that the organism attacked by measles offers less resistance to the intoxication and infection from diphtheria. The aid which a specific treatment usually furnishes in so excellent a manner in overcoming diphtheria is of less value in the case of patients suffering from measles and diphtheria. Even passive immuniza-



tion gives protection but for a very short time. As soon as diphtheria occurs in connection with measles it shows a tendency to wide distribution, to a rapid implication of the larynx, quickly descending into the bronchi.

Wherever I have had an opportunity for a bacteriological examination, all cases of so-called *morbilli-croup*, in which there is an active formation of membranes, have shown, by the presence of diphtheria bacilli, that they were of a diphtheritic nature. In no other case have I seen the propagation of a diphtheria exudate from the tonsils to the bronchi in all its divisions, into all lobes of the lungs, in the course of twenty-four hours, except in a girl aged four years, who upon the day of the maximum in the eruption of measles showed the first coating upon the uvula and upon the next afternoon succumbed, showing a temperature of 107.6° F. Her elder sister who had previously had an attack of measles and at the same time was suffering from a severe attack of diphtheria recovered.

The course of diphtheria in measles is then particularly fulminant and markedly febrile when it occurs at the acme of the period of eruption or just prior to this. If the second infection occurs during a later period in measles it may resemble the course of the usual forms of the disease. Very frequently, in these cases the palate and pharynx remain free from membrane formation, the infection from the onset at once attacking the larynx.

But even then, when measles follows diphtheria with an almost simultaneous infection, both diseases may influence each other in a very ominous manner.

A strong boy aged seven years, in good circumstances, taken ill upon February 18th, from diphtheria, which rapidly assumed large dimensions in the pharynx. On February 20th he received 600 antitoxin units, and on February 21st, after I had visited him for the first time, he at once received 1,500 more. On February 23d he had attacks of fear, without actual symptoms of stenosis, due to marked swelling and formation of membrane in the pharyngeal parts. Moderate albuminuria, then improvement. Upon February 27th, renewed fever. Upon March 2d an eruption of measles appeared, at once severe apathy and high graded asthenia occurred; gallop rhythm. During the night from March 4th to 5th embolism of the left crural artery; absence of pulse up to the inguinal region, severe pain. During the night, from March 8th to 9th, death occurred. In this case the periods of infection with the contagium of measles and diphtheria were close together.

Another very undesirable combination is that of measles and *whooping cough*. As both affections implicate the same mucous membrane areas their influences increase the difficulties and lead to the great danger of severe complications on the part of the lungs and bronchi. It also appears to be the rule here that the combination shows more unfavorable prospects in cases in which the more intense infection—and measles may be properly looked upon as the more severe—is added to the milder, than if whooping cough occurs in connection with measles.

In the former case, particularly in children in the first years of life, an incomplete development of the cutaneous eruption is apt to occur or the eruption is retarded, going hand in hand with a fulminant development of

a very extended bronchiolitis which rapidly leads to death. In the second instance another danger is present, the catarrhal inflammation of the bronchi and lungs, which is due to the measles process, or which has only developed in connection with the appearance of whooping cough, readily takes a sub-acute or chronic character, thus prolonging the total affection for weeks or even for months. Then chronic indurative processes, particularly of the lower lobes, with the formation of cylindrical bronchiectases occur, and then, as well in connection with the local phenomena as with the general condition, with the continued fever and the high graded emaciation, the affection may very closely resemble tuberculous phthisis.

Finally, the connection of *tuberculous infection* with the measles process must be considered. The influence of this acute infection upon tuberculosis is most clearly recognized in those cases in which it attacks the child that suffers from a latent or quiescent tuberculosis of the bronchial glands. Here it awakens to new life—naturally, up to this time in a manner by no means clear—the quiescent germs of the chronic infectious disease. By way of the lymph channels, or from a caseous area of the capsule of the gland, they reach the surrounding areas, and here give rise to a local miliary tuberculosis, which, if the lethal termination occur soon after the onset of measles, brought about by other complications, may be found in the earlier stages or also, upon a rupture into a bronchus, may show recent tuberculous pneumonia.

An example of such connection is furnished by the following case:

Arthur Gr. began to cough at the age of eleven months. Then he had an attack of varicella. In the second half of the twelfth month the cough was aggravated. In January, 1880, a child aged one year, which was very pale and miserable, with a profuse rattling cough, and high irregular fever, but without signs of an implication of the finer bronchi was seen. This condition continued for nearly a month, then the cough disappeared, but later frequently returned. Strength and weight, however, increased in a satisfactory manner. At the beginning of June he was attacked by measles. On June 3d the eruption appeared, a disseminated bronchitis was added, and upon June 8th the child was found dead in bed. **AUTOPSY.** Child still well nourished. In the lower lobes both lungs show short and firm pleuritic adhesions. Left upper and right lower lobe show flaccid infiltrations, beneath which there is a large caseous focus about 1 c.c. in diameter. Right middle lobe shows flaccid infiltration, partly atelectatic. A very large gland at the bifurcation of the trachea shows complete caseation, one half of which is softened. Quite recent development of tubercles in the spleen and in the liver. Mesenteric glands swollen but nowhere caseous.

In another series of cases the first symptoms of a "*scrofulous*" mucous membrane inflammation occur directly in connection with measles, for example, stubborn conjunctivitis palpebrarum, then phlyctena and ulceration of the cornea, with severe eczema about the eyes, the face and the head appear. Now the cervical lymphatics enlarge, cold abscesses develop, and a few weeks later suddenly the neck is seen to swell, and a *tumor albus* develops. Again, a few months later an individual attacked in this manner may develop a tuberculous meningitis and succumb. Here the condition appears to be reversed; measles has attacked a previously healthy child, and only on account of the

acute infection does the individual become favorably influenced for the entrance of the tubercle bacillus; in the exanthem described, the germ may enter injured areas of the skin or the mucous membrane of the nose or eyes. Much more doubtful, and as yet not determined by accurate observation, is the direct infection of the lungs and the bronchial glands by tuberculosis during an attack of measles. The fact must be looked upon with certainty, that not rarely children as well as adults that have been entirely well previous to an infection by measles, show the first signs of a beginning pulmonary affection after the disease has run its course. Among the acute infections, particularly measles with whooping cough takes the first place.

Perhaps the following case of previously diseased lungs, as during an attack of measles, may be looked upon as an immediate tubercular infection.

A child aged two years, that from the end of its first year of life frequently suffered from bronchitis, is admitted to the clinic with the symptoms of a fibroid chronic pneumonia of the left lower lobe. After the onset of phenomena of marked dulness, which remain about the same, so that empyema is constantly suspected, an aspiration proves negative; some time later, the symptoms of cavity formation occur in the affected lobe. Dozens of times the expectoration is examined for tubercle bacilli with negative results. The improvement in the general condition and the increase in weight are opposed to the diagnosis of tuberculosis, therefore, a diagnosis of atrophy of the lung with bronchiectasis is made. When three years of age, upon November 28th, the child had an attack of measles. Readmitted to the clinic upon December 12th, fever returned and emaciation was rapid. Now, to our surprise, the examination of the expectoration showed numerous tubercle bacilli. At the autopsy it was shown that a high-graded bronchiectatic condition, with secondary atrophy of the left lung, was present, because in the markedly swollen bronchial glands, as well as in the healthy lung, a moderate amount of fresh young tubercle nodules were found disseminated. In the preparation which was examined by Virchow a caseous focus could not be discovered.

The possibility of a special liability of the eruptive and convalescent stages of measles (perhaps even of the prior stage) to a tuberculous infection gives a very important direction to practical treatment. The little patients, particularly at this time, must be guarded, with special care, against contact with tuberculous patients.

## DIAGNOSIS

The detection of measles during the time of the eruption is easy in general for the physician who has seen a number of cases. The art of recognizing cutaneous eruptions rapidly and correctly cannot be taught by books, no detailed description, no matter how skilfully the words are chosen, not even the reproduction by means of pictures, but exclusively and alone can this be taught by clinical experience. This is also true of measles. The configuration of the individual points in the eruption, their influence upon the surrounding areas, the distribution over the body are, however, so characteristic that even in the examination of a few cases the well developed eruption may be recognized by the beginner, even by the layman. Nevertheless, marked diagnostic difficulties even occur to the most experienced; perhaps more rarely



in the case of the child than in the adult. Here in some cases the individual macule of measles, particularly in the first appearance on the face, is so markedly infiltrated that a number of distinct but very close nodules upon the forehead, eyelids, cheeks, nose and the surroundings of the mouth, which are intensely red and have a glistening appearance, give such an impression that the experienced observer is more likely to consider a beginning smallpox than an attack of measles. Even to-day there remains in my memory the horror of a very sensitive, nervous musician who, during the time of an epidemic of smallpox, was admitted late at night to the Leipzig Hospital, and placed in the smallpox division, but whom I was able to free from his unpleasant surroundings on the next morning, fortunately without having acquired smallpox in addition to his measles. At another time I observed in a student who had 15 to 20 large bright nodules distributed over his entire body, exactly like a beginning varioloid, the eruption being surrounded by a red areola, that only upon the succeeding day was the first appearance of a very well developed eruption of measles noted in the surrounding of the nodules.

A second difficulty is occasionally due to the differentiation from scarlatina. Sometimes measles—but quite rarely—may retain its original small macular character without the larger serrated figures appearing, the entire eruption then resembling scarlatina. If then, accidentally, in place of the usual catarrh, the pharynx particularly is intensely red or even a lacunar tonsillitis appears, the differential diagnosis may be exceedingly difficult, a condition which I have seen. Then it is always important that the surroundings of the mouth and chin show the same features as the rest of the eruption, these parts remaining free in scarlatina, whereas in measles they are always implicated.

The entirely confluent measles, in which the trunk, upper arm and thigh show themselves as covered by a continuous intense redness, may also confuse a less experienced observer, but the expert will note that in some few completely normal areas, white points in the skin are present in the midst of these red surfaces, this being the appearance in measles, and that if upon pressure over the red areas the small deep reddened points do not return first, that scarlatina is not present. The differentiation may become very difficult in the case of *rudimentary* exanthems, that occur only upon the arms and legs, conditions that happen in both diseases. The exact determination of the accompanying phenomena, particularly those relating to the mucous membranes, must then clear up the situation.

Rötheln in general is of a much lighter color than measles, although the appearance of the individual macules is often very similar, besides fever is usually absent in rubella.

Great similarity is shown by the individual cases of *serum exanthem*, such as we meet with not infrequently in the specific treatment of diphtheria. The previous affection and the absence of the characteristic catarrh lead to the recognition of the proper affection.

The exanthems occurring in the course of other infectious diseases may

resemble measles, for instance in the case of epidemic cerebrospinal meningitis. Quite frequently the "septic" cutaneous eruptions which occur in severe intestinal affections of nurslings show a similarity to measles and may very readily lead to mistakes in diagnosis.

Further, urticaria belongs to the affections which may resemble measles.

Finally, drug eruptions must be considered, for example, those occurring after *antipyryn* and analogous drugs, producing morbilloid eruptions, but care must be exercised not to carry this *finesse* too far. In the instance of a case of whooping cough, in which the child was treated with quinin, a very experienced colleague could not be convinced that an eruption which had appeared was due to measles. As, however, some time later the brother of the patient was attacked with the same eruption, the physician had to admit his error, but the family lost entire confidence in him.

All the previously mentioned difficulties play an important rôle in the case of the individual physician who makes an error, and such mistakes are invariably remembered. But, in general, they are rare and seldom lead to mistakes in treatment, for the careful physician will always delay making a positive decision and will always, under all circumstances, treat the case as an infectious disease.

It is unfortunate that the diagnosis of measles is so uncertain before the appearance of an eruption and prior to the incubation, as well as during the first day of the disease; for during this time the patient is already a menace to those about him, perhaps to the same extent as at the height of the disease, as most contagions occur particularly during the period of invasion, which circumstance we should like to avoid. The appearance of a catarrh, with pains in the eyes and lachrymation, especially in a season in which measles is epidemic, should always be suspicious. The only characteristic symptoms of the prodromal period are the fine spots upon the mucous membranes of the cheeks (Koplik's symptom). In many cases, however, they are not present upon the first day, or are so feebly developed that they are of no value in diagnosis; but upon the second day of the disease they are usually present. In a suspicious coryza we should not omit, at least where we desire to protect other children, to carefully examine the mucous membrane of the cheek. The delicate eruption can only be seen with great difficulty by gaslight. It is best seen by diffuse daylight or in sunlight.

According to my experience, these spots do not occur in cutaneous eruptions resembling measles, particularly in r  theln; they are, therefore, a valuable diagnostic aid for the diagnosis of the stage of invasion.

## PROGNOSIS

Measles represents a comparatively mild, febrile disease, at least regarding the immediate mortality. Hospital experience is capable of showing but insufficient conclusions, as hospitals for contagious diseases as well as children's hospitals in the main admit only severe cases, many of these coming

in after the eruptive stage, and particularly because the division for measles in hospitals constantly shows numerous cases of secondary measles, this always unfavorably influencing the mortality. A much clearer picture regarding general prognosis may be gathered from the experiences of district physicians, those practising in a limited population. Jürgensen,<sup>1</sup> in Tübingen, in 868 cases in twenty years, has observed a mortality of 6.1 per cent.; Fürbringer,<sup>2</sup> in Jena, in one epidemic, only found a mortality of 8.1 per cent. In the district in Leipzig in which I practised for fifteen years, in nearly 600 cases (594) I had a total mortality of 6.5 per cent., therefore, the same mortality as in Tübingen. If it is considered that this character of the disease in the course of two decades has made itself felt in the poor population living under the most unfavorable unhygienic conditions in a large city, it must be admitted that measles is comparatively a benign affection. In hardly any other disease (with the exception of whooping cough) is the course so much affected by the external manner of living of the patient as in measles. In this connection my own statistics are particularly instructive because they may be divided into two periods.

In the first period up till 1884, those portions of the city which composed my district consisted of alleys and cellars and the population was almost exclusively assisted by charity. At that time the mortality was 10.3 per cent. (278 patients). In 1885 the population changed, new streets were broken through, many of the poorest dwellings disappeared and the patients for the most part consisted of better situated workmen and the like. The mortality in the six years of the second period was only 3.1 per cent (316 patients). In the individual epidemic Jürgensen's mortality varied between 3.7 per cent. and 8.9 per cent.; with me in individual years between 0 per cent. (very small number of cases) and 15 per cent. In private practice among the well-to-do the proportion is decidedly more favorable (according to Fürbringer twice as good).

In the individual case the prognosis depends greatly upon the constitution. Secondary measles is particularly serious; constitutions which have suffered on account of malnutrition, bad housing and insufficient care, young children suffering from anemia, severe forms of nephritis, scrofula, etc., always have decidedly poorer chances of withstanding the disease.

Regarding the individual phases of the disease no certain prognostic conclusion can be drawn from the stage of invasion. During the period of eruption a poor development or a retarded state of the exanthem is of unfavorable prognosis as well as the appearance of convulsions; the usual severe cerebral symptoms, somnolence, delirium, etc., are not necessarily unfavorable. In the stage of convalescence any continued or returning rise of temperature after the eruption has disappeared denotes an irregularity in the morbid process.

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<sup>1</sup> "Handbuch der speciellen Pathologie und Therapie," von Nothnagel, Band iv, Thiel 3, Abtheilung 1: "Acute Exantheme," 1895.

<sup>2</sup> Eulenburg's "Real-Encyclopädie," Artikel Masern.



## TREATMENT

When the first case of measles has appeared in a family or has arisen in a school, kindergarten or children's asylum, the question that should be primarily asked is whether and how the dissemination of the disease may be prevented. The endeavors directed to this point are usually made illusory by the circumstance that the diagnosis of the first cases during the first days of the disease cannot be made and, therefore, the surroundings of the first patients are already exposed to the contagion before we are in a position to adopt prophylactic measures. Thus, in an institution, the disease is not limited to a single case, but a larger or greater number follow and the prophylaxis can only then consist in closing the entire institution. In the family, here and there in an existing epidemic, a probable diagnosis may be made in the first hours and then, eventually, other cases may be prevented from arising.

This may be done if the sick child is brought into a special room, particular utensils being used for nutrition and nursing and in having special nurses at hand. Then an isolated service for the patient must be carried out strictly, and all communication between well and sick children, particularly also among the surroundings, for at least three weeks must be absolutely restricted. Under such circumstances I have succeeded sometimes in isolating the individual case. If we are dealing with one or more very young or even weak children the attempt ought to be made to isolate the patient. The circumstance is different if a family is threatened, the members of which have advanced beyond the first years of childhood; in such cases I do not regard a strict isolation as necessary, as measles, particularly during the first years of school life, is combined with comparatively slight risks, as it is very unlikely that an individual child will entirely escape the affection for life. Whereas, in consideration of the same thoughts, the places at which small children collect (day nurseries, kindergartens, asylums, etc.) are to be closed when measles has appeared in these institutions, such a necessity does not exist in the case of schools—except a particularly malignant epidemic is prevalent at the time which would require such a protective measure.

The *treatment* of simple uncomplicated measles may be a purely dietetic one.

It must only be remembered that the disease, on account of a decided implication of large areas of mucous membrane, has a catarrhal character, and that in this respect care is necessary. All possibility of refrigeration, sudden cooling of the skin, wetting without sufficient drying, particularly draughts, are to be avoided. On the other hand, nothing is more important for the favorable course of measles than the entrance of pure fresh air into the sick-room. Both requirements must be carried out by careful nursing, although they appear to conflict with each other.

In persons living in poorer circumstances, even in winter it should be

insisted upon that a window should remain somewhat open in the sick-room day and night. To prevent a threatening draught a curtain may be hung in front of the window and the bed moved away. According to the means of the patient, these conditions can be carried out better and better until finally two rooms may be had for nursing, the one being used during the day, the other at night, so that the one room may be constantly aired while the patient is in the other room. The patients usually ask for the exclusion of daylight on account of the conjunctival catarrh. This request should be granted but without completely darkening the room, which is still done in some families. By the necessary light which is required from time to time, the eye is only more intensely irritated; and, above all, a necessary airing under such circumstances is impossible.

The temperature of the sick-room may be somewhat higher than in other fevers, about 68° F., but not too high, because the dryness which readily occurs under those circumstances directly damages the larynx and trachea. The skin should rather be kept slightly perspiring by the frequent administration of luke-warm drinks (various forms of tea) than be too dry. The necessary washings are always to be carried out with care, member after member being cleansed and very carefully dried. Bathing had better be avoided in measles—in so far as it is not necessary for curative purposes—on account of the possibility of refrigeration. Changing the linen must be carefully performed, this being warmed before being placed upon the child. All these precautionary measures are necessary in the period of convalescence as well as in the preceding period, up to the end of the second week. Except in mild cases the patient should not be allowed to leave his bed before two weeks, preferably not before three weeks; and the patient should stay in the room except in warm sunny weather for at least four weeks. This rule is often broken without consulting the physician and without producing harm; but I know of many cases in which the transgression of this measure has avenged itself, often enough seriously. Even after leaving the bed or the room the regained health remains in an unstable equilibrium for a varying time; great care must be taken to prevent the convalescent from coming in contact with tuberculous patients or their emanations for at least three months.

The nourishment should be fluid during the fever and consist of milk,<sup>1</sup> which, in children that are averse to taking milk, may have a few drops of coffee or cocoa added. As soon as the fever has disappeared and appetite returns, if there is no contraindication on the part of the intestinal canal, wheat bread and butter, meat soups, fruit soups may be given, later vegetables, cooked meat and zwieback, finally, bread and potatoes.

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<sup>1</sup> The advice to restrict the diet to milk, in the fever of measles, to children of the age in which the eruption is met with, is of doubtful value. It is not digested even when relished, in such quantities as are required. Indicanuria at least is a frequent occurrence. The addition of coffee does not improve the taste for children who have been correctly brought up without coffee. The addition to milk should be cereals, on account of their nutritive value and their antifermentative effect.—EDITOR.

After leaving the bed and a few days after leaving the room a cleansing bath is given.<sup>1</sup>

The catarrh of the mucous membranes requires treatment. The eyes are to be washed every morning with boiled water, to loosen the adherent lids, and they are then to be moistened with a salve, such as a zinc salve or a simple glycerin salve; simple catarrhal conjunctivitis needs nothing further.

The coryza requires no other treatment than frequent cleansing of the nose with small swabs of cotton, and protecting the lips by a glycerin salve. This salve is to be recommended on account of its permeating into the upper layers of the epidermis and keeping them moist. By ear specialists (Weiss) it has been lately advised to use a small swab of cotton moistened with a  $\frac{1}{4}$  per cent. silver nitrate solution and introduced several times daily into the nose, the fluid being expelled by slight pressure so that the fluid slowly flows to the choana and the pharyngeal cavity. This is said to prevent infection of the tubes and otitis media.

The oral cavity is also to be cleansed several times daily, in older children by spraying or gargling, in younger children by gently spraying, a measure which is certainly not without influence in the prevention of secondary bronchial and pulmonary affections. If the skin itches it may be anointed with a lanolin salve with the addition of 1.0 per cent. thymol several times daily.

With this dietetic treatment in uncomplicated measles we may get along without a drop of medicine. At most a stubborn cough which prevents the child from sleeping may render necessary the administration of some syrup of ipecac with about 0.005 to 0.02 sulphate of codein to 50 grams of water, a teaspoonful being given three or four times daily.

However, if complications arise the physician must not content himself with this expectant plan but must adopt energetic measures even though here for the most part the physico-dietetic method is also prominent in the treatment.

In excessively severe catarrh of the eyes, after a careful cleansing of the conjunctival sacs several times daily, the palpebral conjunctiva should be painted with an 0.5 per cent. to 1 per cent. silver nitrate solution (followed by cleansing with boiled water); in the interval, under some circumstances, ice treatment is necessary or the application of corrosive sublimate (1 to 4,000) and if the cornea is threatened, repeated dropping of atropin (0.03 to 10). The eyelids are to be rubbed with Pagenstecher's eye-salve (Hydrargyr. oxydat. flavum 1:10 Ungt. paraffin. or Ungt. leniens).

With an intense marked swelling of the mucous membrane and submucosa, with excoriation and profuse secretion, combined with the coryza, the insufflation of Moritz Schmidt's nose powder is to be recommended (three times

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<sup>1</sup> In regard to bathing, which is incidentally recommended (p. 663) in the third week or later, Americans, both professional and lay, have a different opinion and practice. We bathe sooner, on general principles, and to remove the contagion contained in the desquamating epidermis.—EDITOR.



daily): Menthol 0.5, sodium sozodol. 1.0 to 2.0, and 20.0 pulvis sacch. Or the careful pouring into the nasal cavity of luke-warm thymol water  $\frac{1}{2}$  to 1 per cent. by means of a teaspoon (not to be injected!), or the squeezing from a tampon of cotton which has been moistened with a  $\frac{1}{2}$  per cent. solution of silver nitrate. The surroundings of the nose and the upper lip must be protected by the inunction of a covering salve.

Inflammations of the oral cavity, particularly the aphthous forms, besides careful cleansing, require painting with a 2 per cent. to 3 per cent. solution of carbolic acid several times daily. The nurse should be informed that the brush is never to be dipped into the bottle, but a few drops should be poured out into a spoon or bottle or the like and then used.

The first development of noma or cancrum oris is to be treated by an immediate excision of the diseased area, followed by the use of the thermo-cautery. von Ranke has cured three cases in this manner. The condition of the ear requires the greatest attention. It is not necessary after the presence of an otitis media has been discovered by means of the speculum that paracentesis be at once performed. Non-purulent catarrhs of this form recover spontaneously, purulent ones often rupture spontaneously. The introduction of a drop of 5 per cent. to 10 per cent. carbolic glycerin solution and the application of a Priessnitz poultice over the anointed ear are frequently sufficient; but it is necessary to watch the condition carefully as if, with a continued fever upon the first or second day no spontaneous opening of the membrane occurs, paracentesis must be performed. Even afterward the ear must be carefully watched, fresh rises of the temperature, redness, or even pain in the mastoid process, or a possible enlargement of the lymph glands at the anterior or posterior borders of the bone must be looked for so that the diseased portions of the bone and its cavities may be early opened.

The respiratory organs require treatment even more frequently than do the organs of hearing.

In the severe forms of laryngitis in which the submucosa is implicated the methods described below which produce diaphoresis are to be utilized. Local application of heat in the form of poultices, measures which redden the skin, application of mustard plasters, painting with iodine are not to be undervalued. In these cases if we are dealing with somewhat strong individuals, or, better, with constitutions that are not too much weakened, I advise local blood-letting. One or two leeches are applied to the region of the larynx and, according to the condition of strength, the subsequent bleeding is assisted for a longer or shorter time so that 40 to 60 grams of blood are withdrawn. Amelioration always follows this measure and frequently it prevents the development of a serious process. While this measure is being carried out we should never neglect to have the air which is inhaled by the patient in a pure but uniform moist condition (this is best done by repeated spraying at the bedside).

In disease of the bronchi and lungs the various hydrotherapeutic measures are utilized to advantage. In catarrh of the larger bronchi and even in

bronchiolitis, Priessnitz compresses changed three to four times daily are very grateful to the patient. According to the height of the fever, they may even be repeated more frequently and then have a cooling action, if the moistened sheet is not covered with india rubber tissue but only the woolen blanket is laid over it.

In severer cases, measures which redden the skin and diaphoretics are to be used. In cases in which the eruption appears tardily or insufficiently or in which it disappears rapidly, whereas increasing dyspnea, etc., denotes disease of the lung, an artificially produced friction of the cutaneous vessels may bring about improvement. According to the condition and strength of the patient, various methods may be adopted. In very weak children the administration of hot drinks, eventually also of a small dose of pilocarpin<sup>1</sup> internally (5 milligrams to 1 centigram), dry enveloping of the whole body, with the exception of the head, in a woolen blanket are of value, these measures being continued so long as sweating in the face occurs; this is followed by careful loosening of the coverings, thorough drying and rest. In stronger children who can stand some heat and in whom a decided irritation of the skin can be endured, the body is first wrapped in a moist sheet, a woolen blanket being placed over this, and the patient thus enveloped then has another covering placed over these so that sweating occurs as soon as possible.

The "derivative" method is more effectual (in a distributed capillary bronchitis, for instance). This consists in the following: About  $\frac{1}{2}$  kilogram of fresh ground mustard is placed in 1 litre to  $1\frac{1}{2}$  litres of warm water and this is stirred until the oil of mustard develops sufficiently to begin to irritate the eyes and nose. Then a sheet large enough to envelop the entire body is dipped into this mustard water, wrung out and wrapped about the naked body of the child. Or, as in the above case, simple moist linen is used, a woolen blanket being placed over this. This process has the advantage over the mustard bath in that by this method the irritating fumes are not inhaled by the lungs. Where a good reaction has occurred the entire skin of the child up to the neck will be as red as a boiled crab after fifteen to twenty minutes. Now the mustard wrappings are removed and the child is simply enveloped in moist coverings until a good sweating takes place (in from one-half to one hour). The coverings are then removed and the little patient is placed for a short time in a luke-warm bath. If the temperature has risen in the meantime, cold effusions are poured over the chest, head and neck, this being followed by a thorough drying and rest.

This process, at least for the first time, must be superintended by the physician. The last described process is somewhat tedious, but shows surprising

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<sup>1</sup> "In very weak children the administration of . . . a small dose of pilocarpin internally (5 milligrams to 1 centigram) is recommended when the eruption appears tardily or insufficiently or in which it disappears rapidly." One sixth of a grain of pilocarpin is a big dose for a strong adolescent or adult. To give it as above recommended to "weak" children seems to me a mistake. I had an extensive experience with the drug thirty years ago. A strong heart is weakened by it, a weak heart is endangered.—EDITOR.

results; however, it must not be repeated too frequently, at most but once a day, and the child must have complete rest after that for a long time. If after one or two applications the condition of the lungs has not become better nor the eruption more prominent I do not employ the method any further. No good reaction is obtained after the third or fourth attempt and the strength of the child is unnecessarily wasted. However, in cases in which the desired reaction occurs, a repetition upon several succeeding days may be of value.

In place of, or alternating with the derivative diaphoretic procedures, to stimulate inspiration or to bring about deep inspiration which is then followed by strong paroxysms of coughing, cold affusions in the warm bath may be used in the same manner as they are applied in connection with a desired diaphoretic action. According to age, children remain from two to three or five minutes in a warm half bath, of a temperature of 93.5° F., cold affusions (5 to 6 litres in all) being poured over the chest, head and neck. The openings of the ears are to be closed with plugs of cotton. These cold affusions are also of decided use in cases with high fever in which there is marked somnolence, delirium and dry tongue. With a small pulse some alcohol may be given before and after the bath and these baths may be given from four to six times daily.

The action of emetics in bronchitis of the finer tubes is somewhat uncertain. Wine of antimony is best for this purpose, of which a teaspoonful may be given every quarter of an hour until vomiting occurs, but previous to this the child should have a plentiful amount of some warm drink. Local blood-letting in strong children is perhaps more often in place than we are inclined to assume just now. I have had no experience in regard to this. Finally, it is still to be emphasized that in some cases of pulmonary inflammation in measles, with prolonged fever, the so-called antipyretic drugs are of unquestioned value. Here, under some circumstances, they appear to have an antiphlogistic action, in so far as by their administration not only the fever falls but the local phenomena improve and recovery takes place. The following temperature chart (Fig. 24) shows a case of this kind in which antifebrile medication appeared to me to be of decided value.

Thallin<sup>1</sup> was used at that time in the form of injections into the rectum (5 grams of a 5 per cent. solution). Now antipyrin had better be used for the same purpose, 5 centigrams or 2 decigrams daily, according to the age of the patient, or salicylate of sodium 0.1 to 0.5 several times daily, or aspirin in the same dose. It is impossible to say beforehand in which pulmonary complications in measles this medication will be effective. Sometimes it is ineffective, at other times success appears to be marked, but these measures are not to be continued long provided no favorable influence results.

In those cases in measles in which the intestinal phenomena take on a special character, it is valuable, besides the administration of a starchy diet (for example, some of the commercial infant foods), to give for a half or an

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<sup>1</sup>Thallin should remain in its grave. Phenetidin (phenacetin) is preferable to antipyrin, which is liable to cause too much perspiration. It may be combined with caffeine or (when there is any cerebral irritation) spartein sulphate or camphor.—EDITOR.



entire day, every two days, repeated small doses of castor oil (one-half to a teaspoonful) followed by an oily emulsion containing a small quantity of opium. The treatment of nephritis will be described under scarlatina.

Regarding the treatment of sequels and complications, they cannot be considered in detail here. Only two points are to be emphasized. The great danger to the patient suffering from measles complicated by a diphtheritic affection has already been mentioned, therefore, upon the first suspicion the specific treatment for diphtheria is to be used in large and repeated doses (3,000 antitoxin units several times). Besides, it is advisable to at once immunize brothers and sisters of the patient and to repeat this immunization during the duration of the disease and the convalescence (which lasts about six

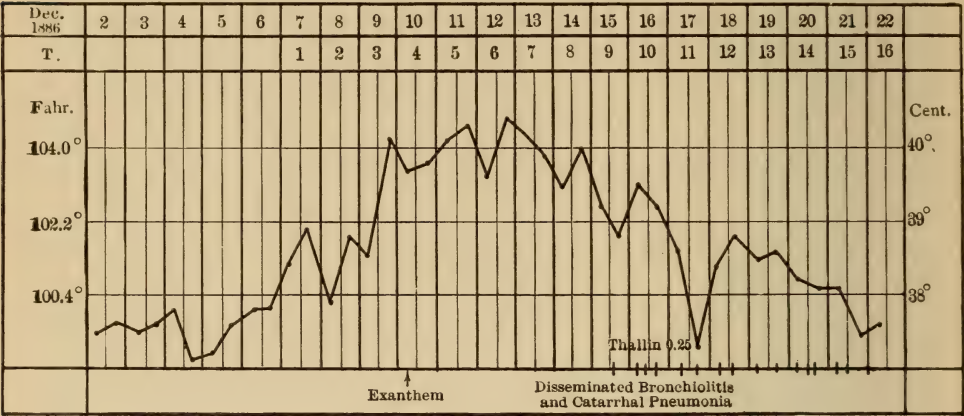


FIG. 24.—TEMPERATURE CHART IN MALIGNANT MEASLES, SHOWING PULMONARY COMPLICATIONS.

weeks) every fourteen days. The same is true of measles occurring in hospitals where the patients cannot be strictly protected from an infection with diphtheria. The experiences in my clinic have shown that by this methodic immunization, diphtheria which was nearly endemic in the measles pavilion has almost entirely disappeared. It is true we had to shorten the intervals between the immunizing injections to less than three weeks, for a number of times severe infections with diphtheria occurred fourteen to sixteen days after the immunization; it thus appears that the antitoxin in the case of measles in particular is rapidly excreted.

Finally, it is to be emphasized again that in all cases of measles where there is a predisposition to tuberculosis (occurrence of a case of tuberculosis in the family) or where local tuberculosis has already occurred (so-called scrofulosis), the convalescents instead of being isolated for but three or four weeks are to be looked upon as half convalescents for several months and as of very unstable health and their resistance must gradually be increased by good nutrition, residence in fresh pure air and exemption from any laborious activity before they again return to the dangers of ordinary life. To recognize the

time when this period has arrived is perhaps possible by an estimation of the body weight, either after the previous weight has been reached and maintained permanently for several weeks or after the child has been brought up to the average weight of its age and maintained at that.

The erection of homes for convalescents in connection with hospitals is an obvious necessity in order to attain this end, especially in case of the poorer classes of the population.

# SCARLET FEVER, SCARLATINA

By O. HEUBNER, BERLIN

## ETIOLOGY

SCARLATINA is an acute, infectious, contagious, febrile disease, characterized by a scarlet-red cutaneous eruption and by inflammation of the pharyngeal organs. The synonyms of the disease in all civilized languages call particular attention to the peculiarity of the eruption.

But little that is certain is known regarding the history of scarlatina. If we reflect how conspicuously the cutaneous coverings of the body are altered by this affection, it does not appear possible that mention of this disease should have been overlooked in the writings of the Arabians and of the ancient physicians. Descriptions from which the disease may be recognized are only found in medical literature in the second quarter of the seventeenth century (by the German physicians, Sennert and Döring). Sydenham in the last quarter of the same century clearly recognized the peculiarities of the disease.

Observations of the disease showed from the onset a very peculiar changeable character in succeeding epidemics, a diversity which hardly occurs in any other affection. Sydenham, in 1664, did not attribute greater importance to the disease than we show nowadays for rubella (rötheln), and fifteen years later, in the sphere of activity of this great observer, in London, the affection appeared with a severity which was only equalled by the bubonic plague. And if one hundred and fifty years later Bretonneau, a physician of like importance, declared that a scarlet fever patient only died when treated incorrectly, a few years later, when in the presence of an epidemic in which a frightful mortality took place, he was compelled to acknowledge how greatly he had been mistaken. An instructive picture of this periodic, very varying course of the affection, occurring in the same population, is presented by a diagrammatic table of the mortality from scarlatina in Hamburg, compiled by Reincke.<sup>1</sup>

The great calamity from which Hamburg suffered in the years 1821, 1831, 1852 and again in 1878 and 1879 from scarlatina is readily recognized, and between these periods there are again very decided diminutions in the danger from scarlatina. The punctuated line added from 1872 on, signifies (in  $\frac{1}{10}$  the scale) the morbidity curve (in so far as it could be determined from the report

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<sup>1</sup> "Die Gesundheitsverhältnisse Hamburgs im neunzehnten Jahrhundert." Hamburg, Sept., 1901, p. 169.



of physicians). It will be noted that the severity of the individual epidemics and the morbidity are by no means parallel. Wherever exact statistical reports have been attained, for example, in Norway, according to the investigations of Johannessen, which cover a period of sixteen years, it is noted that the affection everywhere shows this very remarkable character of rise and fall of the grade of malignancy. In these observations the morbidity and mortality run quite parallel. It is difficult to recognize this property as a consequence of a varying virulence of the pathogenic agent, it is more probable that other auxiliary causes periodically and decidedly increase the susceptibility of the population.

Scarlatina owes its origin to a poison that proliferates anew solely in the human being attacked by scarlatina, whereas outside of the human organism it is capable of maintaining itself in a condition in which it may increase (as a pathogenic organism) but is scarcely ever able to actually proliferate. Previous views, according to which the scarlatinal poison is said to develop in

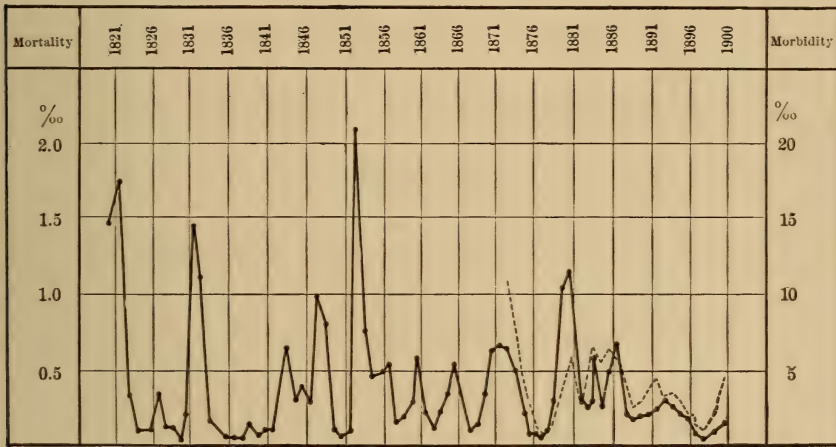


FIG. 25.—MORTALITY OF SCARLET FEVER FROM 1821 TO 1900.

an autochthonous condition outside of the human body, are based upon the observation of cases in districts in which for a long time no case of scarlatina has occurred and also in which no cases were imported. However, since we know that, under some circumstances, the transportation of the poison of the disease may occur by means of healthy individuals, certainly by those that have but a mild degree of tonsillitis, in which the patient does not regard himself as sick, these experiences are no longer tenable with the law of unqualified contagion.

Of this poison, which is renewed year after year in thousands of people, and increases, as little is known regarding its nature as of measles. Since the beginning of the bacteriological era there have been many observers who looked with more or less certainty upon the chains of cocci, which were almost always found in the pharynx of scarlatina patients and very often in the blood and

the tissues of the cadaver, as the pathogenic agents of scarlet fever. The majority of these observers, similar to Sørensen, are probably of the opinion that it is not a parasite which is identical with the streptococcus pyogenes, but that it is a microbe which morphologically resembles the usual streptococcus and one that possesses a special specific character, the property of producing a particular disease. Up to the present, no examination of the streptococcus found in scarlatina has shown the specific property of the germ. Moreover, it has been shown that in all points which can be determined it resembles the streptococcus erysipelatos.<sup>1</sup> In the streptococcus of scarlatinal angina, Hilbert was not even able to determine the property of secreting toxins which Baginsky and Sommerfeld had maintained. The constant occurrence of this microbe in the blood of recent scarlatinal infections (or at the autopsy) could not be demonstrated by Böhm in Baumgarten's Institute, nor by Slawyk in my clinic. Naturally, all tissues were not examined. Perhaps it will be possible by more accurate bacteriologico-biological investigations to solve this question.

[Class recently demonstrated in the secretions of the throat, in the blood and in the desquamating epidermis, a diplococcus which was pathogenic in mice, swine and guinea-pigs.

Baginsky and Sommerfeld have described a streptococcus. Later, Baginsky and Monti showed the presence of streptococci in short and long chains almost constantly associated with other organisms, as pneumococci, staphylococci, and forms of diplococci in smears from the pharynx. The urine, and in two cases the fluid withdrawn by lumbar puncture, contained streptococci. From the autopsy material studied, the streptococcus was isolated in every instance and almost always in pure culture. These observers do not commit themselves in regard to the causal relation of the streptococcus to the disease. The hypothesis that scarlet fever is a streptococcus disease does not receive direct support from recent studies of the blood by Hektoen. This observer made bacteriological examinations during life, especially in regard to general streptococcus infection. He states that streptococci may occasionally be found in the blood in cases of scarlet fever that run a short, mild and uncomplicated course; that streptococci are more frequent in the severe and protracted cases; and that they are not found in some of the fatal cases. Very recently Mallory has demonstrated the presence of very remarkable segmenting bodies in the tissues of cases of scarlet fever.—Ed.]

We are better informed regarding the manner of distribution of the scarlatinal poison than of its nature. Our northern colleagues who have had an opportunity of studying the disease upon isolated islands or in villages situated in the woods in which little communication existed, have been able to supply us with valuable points.<sup>2</sup> Especially the epidemic in Lommedalen, studied by

<sup>1</sup> In 1888 my pupil, S. Lenhartz, succeeded in producing an erysipelatos affection in mice by means of a streptococcus cultivated from scarlatinal blood. (*Jahrb. f. Kinderheilk.*, Bd. xxviii.)

<sup>2</sup> Hoff, "Erfahrungen auf den Faröerinseln über Scharlach." Quoted by Jürgensen, Scharlach in *Nothnagel's Handbuch*, iv, 3, 2. Abth., p. 3.

Johannessen,<sup>1</sup> is particularly instructive, because this author had an opportunity of observing the entire population, amounting to 533 persons, in a village near Christiania, so that not even the mildest case of scarlatinal angina could escape him. From these observations, the individual points of which cannot here be entered upon in detail, it could be seen that nothing but human intercourse, and only this, is capable of distributing scarlet fever. Most frequently it is the mild or not severely affected patients that communicate the disease to susceptible healthy individuals; and the most dangerous transmitters are those adults in whom the disease often exists as an angina (tonsillitis) which is not even accompanied by fever. Thus the entire epidemic, in the previously mentioned lonely Norwegian village, was produced by a servant girl aged twenty-nine who had nursed a scarlet fever patient in Christiania, and being attacked by angina tonsillaris, on the third day of this disease returned to her home situated in the midst of a forest, four hours from Christiania. She was not ill enough to take to bed, but was out daily. She spread the contagion to her own and to other families and from these foci the disease was distributed to over 24 families that lived quite distant from one another, attacking in all 67 persons, or 13.9 per cent. of the entire population, in so far as they were not protected by a previous attack of scarlatina (44 cases).

In large cities, in the greatest majority of cases, the first flaring up of an epidemic occurs by infection in the schools. Those attacked take the disease home with them and transmit it. Among the schools, all those places are to be included in which a large number of children are thrown together: therefore, kindergartens, infant institutions, asylums, etc. Children's parties not infrequently give an opportunity for transmitting the disease. In by far the greatest number of all transmissions, the active agent has naturally not yet been recognized as a scarlatina patient, either because the affection is so mild that there is no thought of a severe foundation of the scarcely noticeable throat difficulty or because the patient is only at the onset of the disease. How frequently does it occur that children, on account of sudden illness and vomiting, are sent home from school, are put to bed and develop scarlatina. During this onset of the disease they have had an opportunity of transmitting the malady to their susceptible neighbors. A second category of infection occurs at the termination of the individual disease. After a fortunate recovery from the affection the little patient is looked upon as completely well if he has regained his former strength, appetite and sleep, and no more of the pathological changes can be found in him. The patient, even as regards his own sensations, has recovered, but in spite of this the poison still adheres to him. It is not to be doubted that this is still the case up to the end of the sixth week after the onset of the disease. A child that had completely recovered from scarlatina, at the end of the sixth week, on account of the still existing discharge from the ear, was sent from my scarlatina division in the hospital to the ear department

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<sup>1</sup> Johannessen, "Gut abgegrenzte Scharlachepidemie in Lommedalen." *Archiv f. Kinderheilk.*, Bd. vi, 1885.



of the Charité. Four days later his neighbor was attacked by scarlatina. Similar experiences in greater numbers may be gathered from the return of children to their own families, that have recovered from scarlet fever in the hospital, even if the proof of a causal connection between a new affection and contagion by means of the convalescent cannot be shown with certainty. Now, the question may even be propounded, whether the sixth week is the final period in which a convalescent is no longer dangerous. Usually the desquamation of the epidermis, which lasts until this time, and may even continue longer, is looked upon as the danger period for infection, and the scales themselves are supposed to be the carriers of the poison. This assumption is by no means proven. It is just as likely that the poison is contained in the pharyngeal organs, in the pus from the ear, in the urine, or in other excretions.

In fact, even of the recent scarlatinal case, it is by no means known with certainty regarding the seat of the contagion, whence it disseminates itself and how it leaves the body so that it is capable of entering a second organism. Clinical experiences render it quite likely that the first point is the mucous membrane of the pharyngeal organs, perhaps, also at least at the onset, this is its principal point of reproduction. The transmission appears in quite a number of cases to occur in a fleeting manner, i. e., without direct corporeal contact, kissing, shaking of hands, etc. The expulsion of sprays of saliva from the mouth of the patient in speaking, which is pointed out by Flügge, may be the method of transmission of the infection. Probably more frequently, especially in the case of younger children, the introduction of the finger or other substances which have come in contact with the poison, into the mouth of the healthy brings about the contagion. This mode of transmission is facilitated by the property of the scarlatinal poison of adhering to fomites, such as toys, books, tools, which a scarlatinal patient has used, for some time, also to letters, linen, beds, clothes, etc. Food substances also, in case they are not heated before use, such as cakes, and especially milk, may retain the scarlatinal contagion. That the poison may remain in rooms, on the wall paper, in hallways, and even apparently after thorough disinfection, has been proven by the frequent unfortunate experiences in cases where children have been away from home and upon returning have been taken sick in a carefully cleaned room. The other possibilities, however, also remain open that the poison may still adhere to the body of the apparent convalescent or perhaps may adhere to the nurses or parents that have shown but a slight attack. That a healthy person, and a healthy third person, may be the carriers of a contagion will probably belong to the rarest possibilities, but that this does occur cannot be doubted. Johannessen communicates an unquestionable case which, in my opinion, shows this mode of transmission. It is that of a servant girl whom he himself had treated two years previously for scarlet fever, and who at the time of transmission did not even show a slight angina. It appears, however, that only such persons are capable of transmitting the disease as remain in the immediate vicinity of the patient, as nurses or servants, and who also come in contact with susceptible healthy persons for a prolonged time.

The point at which the poison is taken up on the part of the attacked individual, as has been mentioned, is most frequently the posterior portion of the naso-pharyngeal space and the oral cavity. Besides this, however, the scarlatinal poison has the peculiar property of utilizing interruptions of continuity in the skin as the ports of entrance to the organism. A varicella pustule which has been scratched, a wound upon the penis in a phimosis operation, an accidental tear of the finger, may be the first points of attack of the scarlatinal infection. Puerperal scarlatina also finds its entrance by way of the smaller or larger injuries upon the genitalia, due to childbirth. In such instances, first the wound assumes an unhealthy appearance, the borders showing a smeary or membranous coating, then the surrounding area becomes red, and thence the general cutaneous eruption begins to develop itself. The circumstance that in the coating of the wounds in such cases, especially in pregnant women, streptococci are always found, Sørensen quotes in proof of his assumption that this streptococcus possesses etiological connection with scarlatina. If only the streptococcus were not the principal factor in most of the other puerperal processes even in those in which scarlatina does not come into question at all!

The susceptibility of the human race at this time is limited; perhaps in this connection not all people and races are alike, even individual families appear to possess absolutely no resistance toward scarlet fever. Under certain external influences (the season) the susceptibility may be increased. But everywhere adults are more resistant than children. Scarlatina, in a much narrower sense than in the case of measles, is a disease of childhood. Johannessen saw in Lommedalen 28.1 per cent. of children, 5.1 per cent. of adults, attacked (in the families in which contagion occurred, 36 per cent. of the children and 83 per cent. of the adults remained free). In childhood the susceptibility is greatest between the third and fourth years of life, but remains high, however, up to the tenth year and then declines. Nurslings are much less susceptible, especially in the first half of the first year of life. I have never seen a positive case of scarlatina in a child under six months of age. (A case recently reported by Kroner,<sup>1</sup> in which a child aged seven weeks was said to be attacked by scarlatina, is somewhat doubtful.)

Recovery from the disease in the greatest majority of cases, confers immunity toward new infection, certainly up to the adult age, when the susceptibility becomes lessened of itself. But the protection against scarlatina appears to me to be slighter than in the case of measles. Among 359 cases treated by me in Leipzig, I met with 6 cases that had been treated for scarlatina, by competent physicians that were known to me, and that only five years previously. Two of the cases were merely attacked by severe angina, while a brother at the same time succumbed to a fatal attack.

*The period of incubation* of scarlatina is liable to greater variations than any of the other acute exanthemata, but in the main it appears to be a short

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<sup>1</sup> *Deutsche med. Wochenschr.*, Nr. 51, p. 896.

one. The well-known case communicated by Trousseau <sup>1</sup>—provided there were no source of error—would show a period of incubation of twenty-four hours, the most usual duration of the period of incubation will be found to be between four and seven days.

### PATHOLOGY

The pathological changes occur particularly in the mucous membrane of the pharynx and in the skin. The changes in the kidneys will be described later on.

In the pharynx, in the milder cases, there is a very decided hyperemia of the mucous membrane and a hyperplastic process in the entire adenoid substance. As well in the larger deposits of this tissue, in the pharyngeal and palatine tonsils, as everywhere in the separated follicles of the mucous membrane, at the base of the tongue, of the lateral and posterior wall and at the entrance of the pharynx, a distinct increase is noted in the lymph cells filling the fine fibre net. The same condition occurs in the lymph glands in this region belonging to the inframaxillary and retropharyngeal region. The sharp limitation of the inflammatory hyperemia of the mucous membrane of the esophagus and entrance to the larynx, which may still be recognized in the cadaver is noteworthy and characteristic. Particularly in the first-mentioned area, the hyperemia terminates in an abrupt straight line. The upper surface of the mucous membrane is found to be in a catarrhal condition with a mucopurulent exudation, especially upon the surface of the tonsils.

In the severe cases, which terminate fatally in from one to four days, this acute hyperplasia of the lymphatic tissue is often found developed to an enormous extent over the entire body. Not only the peripheral lymph glands upon the neck, those in the axilla and in the inguinal region enlarge, but also those throughout the entire intestinal tract, as well as all the lymph follicles, all Peyer's patches, the mesenteric glands, and even the spleen are enlarged to a similar extent; upon section this latter organ shows medullary swelling, it is pulpy and coarse. New formations of small lymph nodes are found in the liver and in the kidney. The last-mentioned organs in these cases appear markedly hyperemic, here and there small hemorrhages are found, particularly near the surface. According to van den Berg,<sup>2</sup> the blood with but few exceptions shows a hyperleukocytosis which lasts for several weeks and is due to the polynuclear elements. The heart in the rapidly fatal cases is usually flaccid and pale; but also quite firm and markedly contracted left ventricles are noted.

In cases of a slight or medium severe infection, the more locally limited inflammatory disease of the mucous membranes and lymph gland enlargement diminishes in the second half of the first week or in the beginning of the second week, even though, subsequently, in one or the other gland, a more

<sup>1</sup> *Med. Klinik des Hôtel Dieu*, 2te. Aufl., German by Culmann, Bd. i, p. 98.

<sup>2</sup> *Arch. f. Kinderheilk.*, Bd. xxv.



substantive inflammatory process may develop. In very many cases, however, the serious turn which the affection takes occurs in the pharyngeal organs. The simple severe inflammation then changes its character and leads to an inflammatory necrosis of the tissue, by the formation of a coagulated, simultaneously hemorrhagic, exudate. Partly (but usually not to a marked extent) this is found upon the surface of the mucous membrane, between and beneath the epithelium, in the form of thin, often disconnected membranes, partly to a slighter, at other times to a more marked extent, in the tissue of the mucous membrane and in the adenoid layers. But even there this inflammation (in an anatomical sense a true diphtheritic one) does not halt, but attacks the mucous gland layer situated beneath the mucous membrane, as well as the fat and muscular tissue, for example, of the palatine arch and uvula, of the epiglottis and of the larynx, altering everything into a rigid opaque mass, of which the original morphologic composition is no longer discernible. The inevitable result of this "coagulation necrosis" as it has been very aptly designated by Weigert, is the destruction of all affected parts. This inflammatory necrosis also attacks deeper lying organs, especially the lymph glands situated near the pharyngeal parts, where it leads to the development of gangrenous hemorrhagic foci, which again are a source of danger to the surrounding areas, the cellular tissue of the throat. This tissue shows phlegmonous inflammation with widely distributed rigid tissue infiltration, with the formation of gangrenous foci varying in size. In the internal organs, especially the liver, circumscribed diphtheritic foci of slight extent may develop.

It is remarkable that in this diphtheritic, or better—to differentiate the process from genuine diphtheria, with which etiologically it has nothing in common—*diphtheroid inflammation* of the pharyngeal organs, with extremely rare exceptions, in the cases that die during the first days of the disease, even though the adenoid conglomerations are as markedly hyperlastic as possible, nothing is found. The starting point appears to occur mostly upon the fourth day of the disease.

The following case is an illustration of a beginning diphtheroid condition which has just been described.

Swob., Albert, aged ten years, attacked upon June 13, 1888, in the afternoon, with high fever, limited eruption, delirium, unconscious upon the 16th, and died upon the afternoon of the 17th of June.

The pharyngeal parts show a line of demarcation at the velum palatinum downward to the entrance of the esophagus, consisting of a sharp border of a dark bluish-red color. Inside of this area, upon the uvula, fine, hemorrhagic, punctiform regions may be noted. Upon the surface and in the lacuna of the left tonsil there is a muco-purulent coating. A section of the tonsil shows a marked medullary, swollen consistence of a reddish-white color. The same condition is noted upon section of the right tonsil, in its lower half. The upper half, however, shows a sharp demarcation compared with the lower, consisting of a high-graded, dark red discoloration, due to an inflammatory, hemorrhagic infiltration. A stream of water let fall upon it shows that the upper surfaces of the tonsillar swelling in these areas have separated and that a necrotic decomposition is beginning.

This diphtheroid process is combined with a very regular bacteriologic finding which is so constant that we may speak of a relation of the one to the other. Whereas in the first days it is impossible to find microbes in the microscopic sections, even in the markedly swollen tissues—their presence very frequently may be determined bacteriologically—in the diphtheroid areas, great masses of streptococci are found, and not only this, they are found in the spaces of the tonsillar tissue and may even quite often be found in sections in the tissue of the lymph glands.

Regarding the rôle which is to be attributed to the streptococci in this dangerous process, the opinions of those authors who deny an etiological connection between streptococci and the scarlatinal poison are still divided. Some look upon the serious change which occurs in the scarlatinal mucous membrane as the direct action of the previously mentioned microbes, which, already present to a lesser or greater extent in the oral cavity, find opportunity to gain a firm foothold upon the changed mucous membrane areas due to the scarlatinal infection, and, entering the tissue, produce an inflammatory necrosis. Others believe that the scarlet fever diphtheroid is due to the action of the still unknown scarlatinal virus itself in especially susceptible individuals that have a weak mucous membrane, and only the necrosed tissue itself opens the tract for the massive proliferation of the cocci chains and their entrance into the organism.

I favor the second view in regard to the circumstance that the diphtheroid mucous membrane disease—therefore, the beginning of the tissue necrosis, not the surface exudate!—so commonly holds to a distinct phase of the course of scarlet fever and that streptococci in every case of scarlatina are present in the pharynx, diphtheroid, however, not always taking place. A decision will only be possible after we have learned to recognize the scarlatinal virus with certainty. Practically of great importance, however, is this, that the streptococci after they have found an easy entrance into the organism affected by scarlatina become very potent in regard to the further course of the affection. The opponents of the law of the specificity of the streptococci must also admit that the resistance of the organ to the deleterious effects of the microbes in scarlatina is particularly slight, less than in almost all other diseases. For this reason, streptococcus sepsis plays such an ominous rôle in the prognosis of scarlet fever. At one time this shows itself in the form of severe phlegmonous inflammation of the throat, at other times the coccus infection is transmitted by way of the lymph tracts, again it creeps along the retrotracheal and mediastinal connective tissue tracts, at other times the coccus infection enters a vein and produces secondary pyemic blood disintegration, with multiple, purulent metastases in the most varied parts of the body. Scarlet fever pyemia, deviating from other analogous infections, shows the peculiar character that the joint cavities and the serous membranes are particularly predisposed to purulent deposits. In all cases of this kind we may speak of a proliferation of the streptococci through the entire body and in such quantities, very much greater than the relatively distributed germs

which are found in recent scarlatina cases (without diphtheroid) even in the first days of the disease in the blood. The amount of streptococci in the blood, according to the investigations of Slawyk,<sup>1</sup> who worked in my clinic, observing all necessary precautions, in general is parallel to the intensity of the diphtheroid mucous membrane inflammation, even if this is not the only point of entrance for the microbes, but may share this property with other inflammatory mucous membrane diseases, especially of the auditory canal.

Slawyk found the blood, in 15 cases which terminated fatally on the third day of the disease, constantly sterile, only upon the fifth day of the disease the investigated cases showed, and then in decided quantities, streptococci in the blood.

If it is true that streptococci do not produce toxin formation, it must be assumed that the bacteria themselves, by their property of producing inflammation, give rise to the severe, mostly fatal affections. However, we have observed a number of cases in which, in spite of septic fever and of the fact that the other clinical phenomena pointed to sepsis (such as the gradually decreasing power of the heart, the loss of appetite, emaciation, the frequent implication of the sensorium) neither purulent foci in the body nor streptococci in the blood could be determined, therefore, death appeared to be due to a pure septemia. These were mostly cases in which stubborn and severe otitis media complicated the disease (but without sinus thrombosis or other local infection of neighboring organs). Observations of this kind require further bacteriological study.

The *changes in the skin* in scarlatina require further pathologico-anatomical study. The investigations up to the present do not favor a simple "vasomotor" hyperemia, such as is produced by chemical poisons, but they favor inflammatory processes. Besides marked hyperemia of the papillary body, the vascular nets in the deeper layers of the cutis are greatly dilated and markedly filled with blood, and along the smallest veins narrower or broader streaks of round cells are noted, which cannot be looked upon as anything else than as emigrated leukocytes. With this, the tissue of the cutis and the cells of the rete Malpighi are apparently richer in fluid, as if swollen, this may even be noted in hardened preparations. The fine dark points which we shall learn to recognize in the description of the eruption, apparently lie in the deeper layers of the cutis (third venous net of Spalteholz),<sup>2</sup> whereas the general redness may be ascribed to the papillary body.

## SYMPTOMS

Our description shall represent the picture of an ordinary, medium severe attack of scarlatina. It corresponds to a certain type which this acute exanthem possesses, although at the bedside deviations from this model are in the majority.

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<sup>1</sup> *Jahrbuch für Kinderheilkunde*, Bd. liii, Heft 5.

<sup>2</sup> "Die Vertheilung der Blutgefäße in der Haut." *Arch. f. Anat. u. Physiol.*, 1893.



In rare cases indistinct phenomena, general disturbance in health, often chilliness and the like, may precede the disease for a few days and even for a week, which may be looked upon as symptoms of the period of incubation. Usually, however, the disease begins abruptly with severe symptoms, so that the chronology of the affection may be accurately determined by the hour. The child returns from school in complete health, a quarter of an hour later vomiting occurs and at once high fever appears. Or it has gone to school quite well and there has an attack of nausea, vomiting appears and the disease begins.

The most frequent symptom at the onset is *vomiting*, according to the time at which the last meal has preceded, either remains of nourishment or of mucous fluid, or sometimes even bile-stained masses, are brought up. In younger children diarrhea is readily added and then lasts for a day or two. Even in nurslings, vomiting and diarrhea are the earliest symptoms.

In cases of medium severity the vomiting may be repeated several times during the first hours of the disease and then cease. Immediately following this a general febrile condition appears. In older children, it may be introduced by a true chill, also in younger ones (rarely) although not as an initial symptom; also, inside of the first few hours or first two days, general convulsions occur, which are not always of unfavorable prognosis. A general feeling of malaise, *headache* (also pain in the limbs), lassitude, irritability, pains in the abdomen always rapidly follow. Somewhat later, from twelve to twenty-four hours after the onset the child complains of its throat, of pain in deglutition. In adults, on the contrary, the pain in the throat is frequently the first symptom.

In practice, not infrequently, there is an opportunity of examining the child shortly after the initial vomiting. It is always found with marked fever, but not always is this high at the onset, but varies between 102.2° F. and 103.1° F., but, occasionally, even in moderate cases, the temperature rises at once to 104° F. to 105° F. (in the rectum). The child assumes the recumbent posture itself, and now a febrile condition is noted which lasts from eight to ten days. Even during the first night, febrile unrest, mild delirium appear, while during the day the child is apathetic, sleepy, complains of thirst, burning and pain in the throat, the speech assumes a muffled, throaty character.

Examination of the oral cavity shows a tongue with a white coating, the filiform papillæ are somewhat reddened and the palatine mucous membrane, with a quite sharp line of demarcation toward the hard palate, is markedly reddened, showing distinct small maculæ, the tonsils are swollen and upon their deep reddened surface, not infrequently, reddish, yellowish flakes or striæ may already be noted.

The face sometimes appears bloated, in some cases transversely over the nose a saddle-like redness is noted, which coalesces with the fever redness of the cheeks, whereas the surroundings of the mouth and chin are almost unnaturally sharply demarcated by a conspicuous pallor.

In the course of the first day or in the first half of the second day the characteristic cutaneous eruption appears, usually first upon the trunk, neck, chest or back, also in the gluteal region and then, gradually, in the course of two days, distributes itself to the extremities. The characteristics of this eruption consist in two peculiarities: At the onset it consists of small points distinctly separated from one another, which in the course of hours or days coalesce, forming a uniform connected mass of a red appearance and, secondly, at the onset it is of a delicate red, rapidly or also slowly, even in the course of several days, assuming a saturated, burning, flaming, fire-red appearance which, therefore, justifies the name of scarlet fever. In all cases that are at all developed, this general redness, no matter how intense and flaming it becomes, attains its development from separated punctiform areas in so far as in the general redness, upon accurate observation, small still darker flakes may be recognized, especially if by means of slight pressure upon the skin the blood is forced away. Primarily the redness upon decreasing pressure always appears first in the original red areas.

Therefore, upon the neck, on the chest, upon the back, upon a delicate white skin, there are first noted delicate rose-red punctiform, from a millet seed to a pin head in size, particularly round, areas situated close together: At a distance a uniform color is presented, and only upon close observation can the individual points be discerned. Soon the entire trunk appears to be covered and now, at the end of the second or the beginning of the third day, upon the inner surface of the arms, the thighs, soon also taking in the outer surface, reaching to the fingers and toes, the same fine redness is noted. The skin at first feels smooth, but with increasing intensity of the redness it becomes uneven, coarse, something like shagreen leather. The face frequently remains entirely free or only signs of the fine red points are noted upon the temples, the dorsum of the nose or on the outer surface of the cheeks. The region about the mouth and chin invariably remains free. With each succeeding half day the red points coalesce more and more and become darker, upon the third, fourth, sometimes only upon the fifth day, the skin from the throat to the feet shows a condition as if the patient were covered by a scarlet mantle which closely envelops the entire body. In the inguinal region, at the elbow, and at the knee joint, upon the buttocks, upon the inner surface of the thighs, the eruption is usually particularly intense, often showing a bluish-red shade. It is not infrequent, even in cases running a normal course, that in these previously mentioned areas the original red points show a hemorrhagic tendency and are then particularly noticeable.

In not a few cases, particularly upon the abdomen, but even upon the lateral aspect of the thorax, upon the back, upon the lower legs, the dorsum of the hands and feet, over these original spots there appear vesicles the size of a millet seed, filled with a transparent fluid, which later becomes opaque; these are called miliaria, which after a few days dry and form scales. The contents of these vesicles show an alkaline reaction, therefore, do not consist of the secretion of the sweat glands alone. This form of the eruption

is called *scarlatina miliaris*, and its appearance is not considered of unfavorable prognosis; in fact, most cases of *scarlatina miliaris* run a favorable course.

Opposed to this—but even this again has exceptions—is another deviation of the exanthem. Upon the third or fourth day in the occasionally slightly uneven, but not particularly raised, eruption, nodules and papules the size of a lentil seed, usually of a deeply stained color, are noted to appear, which are situated at some distance from one another and sometimes produce great itching. In those cases in which the rash has a limited distribution these papules may end the general redness and then show an appearance which simulates urticaria. This form of the exanthem is seen much more frequently in the severer infections.

Upon the dorsum of the hands and feet the skin occasionally swells and becomes glistening, sometimes almost resembling erysipelas.

A peculiar condition is noted in the fully developed exanthem if the finger nail is passed over the red surface, in the entire extent over which the nail has passed a white streak of varying intensity is noted (apparently due to vaso-motor spasm) which only slowly disappears (*raie blanche* of the French).

After the greatest intensity has been reached the eruption remains at its acme for half a day or longer, then it begins to fade and disappears at the end of the first week or the beginning of the second, frequently slight exacerbations being noted in the interval, these usually occurring toward evening. Even before the fading is completed the epidermis begins to peel in many areas, first, usually, upon the neck, and this desquamation continues often for many weeks, even in convalescence. Upon the neck, upon the chest, and in the inguinal region, this desquamation occurs in small flakes or plaques, from the size of the head of a pin to that of a lentil; they are of a white glistening appearance. Upon the buttocks, the thighs, the hands, and feet, however, large lamellæ, and entire casts of the fingers, or the hands, which may renew themselves several times, may be shed. Finally, after a prolonged period of desquamation, the patient enters upon convalescence with a renewed skin.

Occasionally, in this rapid transformation process of the skin, an improvement in chronic difficulties occurs: Thus, in a case of intense desquamation, I once saw numerous warts covering both hands of a seven-year-old girl disappear without leaving a trace.

Hand in hand with the development of the exanthem the fever proceeds. Simultaneously, or even earlier, often even upon the second or third day it reaches its acme, the absolute height of which in ordinary cases may be quite high, reaching 105° F. or even higher. Usually in a favorable course it does not long remain at this height but soon declines, however, not to fall suddenly and rapidly but quite gradually and slowly, so that in the course of a few days, falling a little each day, normal ranges are reached. Characteristic of an undisturbed defervescence is the fact that after the temperature has begun



to decline no increased rises are noted over the previous evening or morning temperatures, whereas in the course of a day the variation between morning and evening still remains. The decline of the fever may be likened to a staircase which gradually leads downward.

The following temperature charts (Figs. 26 and 27) are examples of simple, uncomplicated scarlatina (rectal temperatures). In the first instance the acme was reached rather late, the defervescence being somewhat more marked, in the second case the acme is noted on the second day, the defervescence being slow and gradual. Both cases entered upon an undisturbed convalescence without complications.

The *pulse* in scarlatina shows peculiarities in being very rapid, greater in proportion to the height of the temperature and age of the patient than in other diseases. This is also true of the uncomplicated cases and it cannot even be said that this symptom is more markedly developed in the severer infections. In children from five to six years of age, in the first few days, even in mild cases with a rectal temperature of 103° F., from 150 to 170 pulse

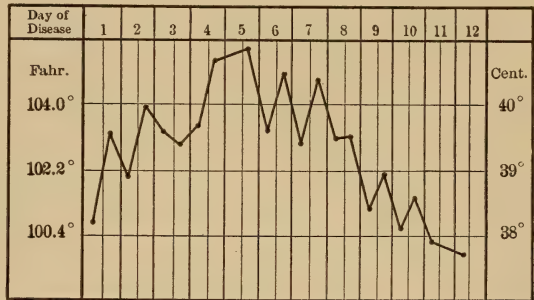


FIG. 26.—TEMPERATURE CHART OF SIMPLE UNCOMPLICATED SCARLATINA.

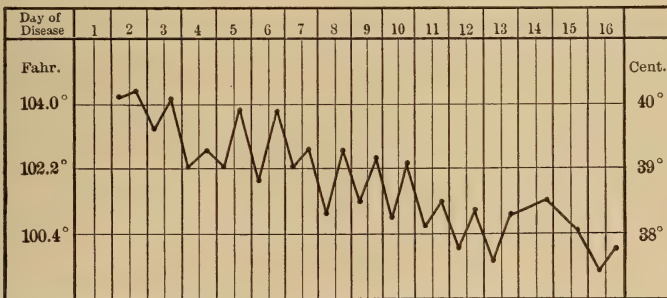


FIG. 27.—TEMPERATURE CHART OF SIMPLE UNCOMPLICATED SCARLATINA.

beats per minute, with a temperature of 102° F., 132 per minute, with a temperature of 100.5° F., 124 per minute, etc. In a girl aged seven (medium severe case), I found, with a temperature of 101° F., 150 beats per minute. With higher temperatures, pulse beats of nearly 200 per minute can be counted (for example, in a girl aged five with a temperature of 104° F.), without the prognosis necessarily being unfavorable. Nor does the pulse rise much higher in the very severe, fatal cases with excessive high tempera-

tures. In a boy aged five, with a temperature of  $107.8^{\circ}$  F. I noted 196 beats per minute. In a case which terminated fatally in a girl aged two, with a temperature of  $104^{\circ}$  F., 204 beats per minute. From this symptom it may be quite properly concluded that the scarlatinal poison, more decidedly than other infectious products, has a direct action upon the cardiac nerves which increase the heart beat. The condition of the pulse in the first days is sometimes rapid, usually, however, it is soft, not dicrotic. Some sphygmographic tracings appear to favor the view that the vasomotors of the peripheral vessels are in a certain spasmodic condition, which is also indicated by the previously-described "white streak." While the exanthem is reaching its full development, the inflammatory affection of the pharynx and the oral cavity increases. The tonsils become more swollen, the muco-purulent or fibrinous flaky deposits upon their surfaces become distinct, but if they were previously present, they now increase and enlarge, the lymph glands at the angle of the lower jaw enlarge and become painful, the tongue loses its white coating and the superficial layer is denuded of its epithelium so that the tongue looks red and smooth, but the papillæ stand out prominently above the surface like red warts. If the child protrudes the tip of the tongue between the lips it resembles a strawberry. A tendency to dryness of the tongue readily occurs upon the days when high fever is present.

With the fading of the eruption and the onset of defervescence the mucous membrane affection improves, the yellow spots upon the tonsils disappear; the tonsils are less swollen, the glands decline in size, the tongue regains its normal epithelial covering.

Other decided deviations on the part of the internal organs are not noted during the acme of simple scarlatina. The respiratory apparatus is hardly affected, at most there may be a slight tracheo-bronchitis. The urine shows the characters of febrile urine, it is scant, high colored and, with an intense fever, may show small amounts of albumin and some few hyaline casts; this disappears as the fever declines. A slight increase in diacetic acid is often noted, the diazo-reaction is negative. The spleen is very commonly enlarged, which may be shown by percussion but may also be noted upon palpation.

Anorexia is present during the entire course of the fever, but there is great thirst. After the preliminary diarrhea the bowels are usually constipated. With defervescence and a decline in the eruption, there is a return of the normal epithelial coating of the tongue, appetite returns, and the digestive apparatus begins to functionate normally.

Toward the middle of the second week the pathological disturbances have almost disappeared, only the skin which has been deeply affected still requires a longer time to regenerate itself and to return to its normal condition.

Strength and subjective well being usually return rapidly. Only the impossibility to foresee the later implication of the kidney influences the careful physician, even in these cases, to keep the patient in bed at least till the end of the third week and to watch him carefully.

## VARIATIONS FROM THE COMMON TYPE

We shall now devote our attention to the *manifold deviations* from the just described simple course, a review of them being only possible in that the individual course is viewed particularly in regard to the organic disturbances which become prominent. Naturally, it must not be forgotten that in each special case these deviations are not always so distinctly separated from one another as they are in text-book descriptions, the disturbances commonly combining, crossing or following one another.

In scarlatina, which quite properly is so much feared, we also note *mild* and *very mild affections*. In such cases all symptoms of the disease are but slightly developed and of a very benign character. At the onset vomiting and headache may be present but the pharyngeal affection is only noted by slight swelling and reddening, the fever rises to a moderate height for but a short time and may sometimes show a purely afebrile character, in other cases only subfebrile rises occur, the temperature only rising to 101.5° F. and rapidly declining, the fever may even sometimes be absent entirely. The eruption consists in a uniform reddening of a pale rose color, distributed over the entire body, which may in individual areas, upon the neck, upon the buttocks, show a slightly raised character or this may be absent entirely; or the eruption may only be found in individual areas, for example, upon the groins, at the flexures of the knee, upon the neck or upon the back, being even fleeting here or remaining for several days. The diagnosis in these cases can only be made with certainty upon a careful observation of the entire body and only then by the aid of such knowledge as that other children in the house have been attacked in a well developed manner or in the presence of an epidemic. Sometimes the eruption is well marked, but only upon one part of the body, for example upon the legs, at other times days pass until the rash distributes itself from one part of the body to another.

The *rudimentary cases* must not be placed in the same category with these mild cases. These at the onset may appear quite mild and many of them even remain so, but very frequently in the wake of these apparently harmless disturbances is a late renal affection. The rudimentary character is shown in that the infection appears in the form of an angina frequently severe, and accompanied by fever, and without any eruption. Especially adults, but also older and younger children, for example, also those that have previously had an attack of scarlatina and have been exposed anew to a marked family endemic are not infrequently attacked in this manner. There is marked fever, often also vomiting and headache, a pharyngeal inflammation, with enlargement of the tonsils and the adjacent lymph glands, often with flaky deposits lasting for some days and then rapidly disappearing and healing without serious consequences, so that the patients appear cured in the course of a few days. Frequently the sharply defined border of the dusky, sometimes even flaky, redness about the hard palate shows the true character of the affection,



at other times attention is directed to the condition by an unusual implication of the nasal mucous membrane, in which there is thin mucoid corrosive fluid which collects about the openings of the nose and the upper lip, producing excoriation; often even these symptoms are absent in the clinical picture and it is thought that an ordinary tonsillitis is present, provided no new unquestioned cases of scarlatina are brought about by such cases or unless an edema develops two or three weeks later and shows the true nature of this angina.

Compared to the interesting pictures of the disease which we have just described, the affection shows itself sometimes in a dreadful, almost demoniacal form, when it appears as a so-called *scarlatina fulminans*, *scarlatina gravissima*, *intoxication form of scarlatina*. Almost like lightning, or like a poisoned arrow, in the midst of blooming health, and in the course of a few hours, destruction has been wrought by this property of the scarlatinal poison, which, in common with some other infections (variola, cholera, bubonic plague, etc.), and in some epidemics, is characterized by a great number of such rapidly fatal cases. In the severest cases the affection up to the time of death lasts from thirty to thirty-six hours. Such cases are fortunately very rare; I have only seen two or three among many hundreds of cases. The severest injury to the nervous system on the one hand, to the heart on the other, often without the appearance of the usual characteristic symptoms, destroys life. The following observation is an example:

Girl aged three and a half years, taken ill with vomiting, in the afternoon of December 6, 1875. An evening temperature of 102.6° F., pulse 176. December 7th, early in the morning, a temperature of 104.9° F., pulse 218, unconscious at noon. At 2.30 in the afternoon I saw the child. There was complete coma, pallor and wide and unreacting pupils. Upon the body an erythema, no characteristic eruption was noted (a brother aged eight simultaneously had a characteristic scarlatina). Temperature 108° F., pulse could not be felt, 196 cardiac contractions. A bath at 68° F. for fifteen minutes, with ice water affusions, followed by enveloping in woolen covers was ordered. Pupils then reacted somewhat but no signs of returning consciousness. Four o'clock in the afternoon, temperature 102.9° F., 108 cardiac contractions. Bath at 77° F., followed by almost no reaction; soon afterward death occurred. The entire affection lasted twenty-eight hours.

By far the greatest number of the cases of this group last longer, from three to four days, until death occurs which, according to my experience, cannot be prevented. The onset of the disease may be fulminant, characterized by very severe and almost continuous vomiting and retching, or also by spasms, and even general convulsions. At other times these serious symptoms only develop upon the second day after a moderate onset; above all, they consist of an unusually severe implication on the part of the nervous system. Sometimes violent, at other times quiet but continued delirium or maniacal irritability introduce the drama and rapidly lead to a profound implication of the sensorium, to complete bewilderment or deep coma. The face usually assumes a peculiar disturbed, frightened expression. This severe implication is usually paired with great restlessness of the motor organ; it appears as if

an internal fear compels the little sufferer to a continuous change of position of the body and thus the patient is often found with the head hanging out of bed, lying across the bed or in a complete inverted position in the bed. Very frequently the respiration is loud, resembling that of a person who has run for a long time or it may be that form of respiration which occurs in diabetic coma, deep, long-drawn and difficult. The appearance of the rash varies greatly. The face is sometimes peculiarly streaked, red, white, pale about the mouth, sometimes there are red streaks upon the cheek, and temple, sometimes dark disseminated, papulous eruptions may be seen, the face as well as the trunk showing a bluish marbling, and only toward the end of life does the rash become more distinct. In other cases, on the contrary, a very rapidly developing intense eruption is noted, which very soon takes on a deep, dark, cyanotic discoloration. In one of my recent cases, in a child aged one year and a half the entire skin of the body was covered by dark blue maculæ, varying in size from a lentil to a silver dollar, which did not disappear upon pressure, while only in some few areas were they smaller, showing a more elevated appearance. The child was not placed in the scarlet fever division of the hospital as its disease was looked upon as a "septic" skin eruption. The pharynx is sometimes but slightly affected, at other times it is markedly implicated, reddened and swollen. The tongue is dry, red, fuliginous, the breath distributes a sweet smell resembling acetone. In the urine a marked diazo-reaction is present, occasionally moderate amounts of albumin and hyalin casts may be noted. The urine as well as the feces are voided involuntarily; the stools show for the most part a diarrhetic character.

The fever is high from the onset or at least from the second day on, not infrequently it is excessive. The pulse becomes exceedingly rapid, often being irregular. Acute dilatation may be recognized in the heart, and the weakness of this organ is the immediate cause of a rapid, fatal termination. In another series of cases this does not occur in such a stormy manner as has just been described, but death takes place unexpectedly by a rapidly appearing cardiac collapse. In such cases there is also high fever, delirium, marked eruption, rapid pulse, but in the interval the child shows freer moments, takes nourishment, being less excitable and more quiet, until suddenly, with high fever, occasionally with slight twitchings (once I saw these twitchings occurring bilaterally), death comes on quickly. In cases of this kind the total duration of the disease is from three to four days.

In a third category of these severest cases, the infection lasts longer, up to six, seven, even to eight or nine days. To the severe general symptoms an extremely high-graded implication of the pharyngeal region is superadded. Whereas the nervous system is not so markedly affected, so that consciousness toward the end of life, even though disturbed, is not completely lost, there rapidly occur difficulties in deglutition and respiration, a true "narrowing of the fauces," due to a high-graded swelling of the entire naso-pharyngeal ring. Speech is throaty, can scarcely be understood, respiration is stertorous, the

head and neck are held stiff, somewhat posteriorly, the entire neck being rigid and immovable. Inspection of the throat shows marked swelling at the base of the tongue, the tonsils, of the palatine arches and of the uvula, so that these structures appear as if joined, and from the fourth or the fifth day on a board-like swelling in the region of both angles of the jaw is added which distributes itself rapidly around the inferior maxilla and, meeting the swelling

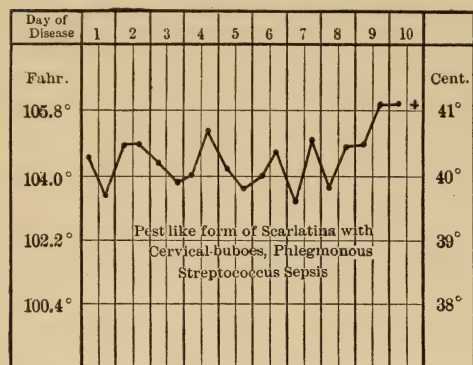


FIG. 28.—TEMPERATURE CHART OF PESTLIKE FORM OF SCARLATINA WITH BUBOES.

subject to this diphtheroid condition, will take place and a flooding of the entire blood with streptococci with subsequent pyemic suppuration will develop. A severe, usually continuous, fever, accompanies this process which already belongs to the realm of sepsis. The course of such a case is shown by the accompanying temperature curve (Fig. 28).

These forms of the disease, with their rapidly developing buboes about the throat, with subsequent phlegmons and widely distributed tissue necrosis may readily be designated as simulating the plague.

They form the transition to *scarlatinal diphtheroid*, the second variety of severe *scarlatinal infection*, the more or less prominent predominance of the condition giving the character to the scarlatinal epidemic. In this variety, disease of the pharyngeal structures and the adjacent organs is most pronounced in the pathologic process and only by this means do secondary general disturbances occur which again may bring about a fatal change in the disease, but then no longer being part of the original scarlatinal process but to a great extent being of a septic nature.

The pathologico-anatomical process which in this variety of scarlatinal infection attacks the pharyngeal organs has been accurately described previously in this article. It need only be repeated that it consists in a peculiar combination of inflammation and necrosis, in which the latter must infallibly follow the former provided a coagulable exudate is deposited in the tissues of the affected organ. This occurs in scarlatinal diphtheroid to a greater or to a lesser extent.

coming from the opposite side, takes in the entire upper region of the throat so that these structures appear to be girdled with rigid armor, rendering movement entirely impossible. This swelling is due to a phlegmonous condition of the connective tissue of the throat in the region of the rapidly infected and gangrenous lymph glands coming from the pharynx. If children remain alive up to the beginning of the second week marked destruction of the organs of the throat, which have been



The disease frequently is accompanied from the onset with high fever and marked swelling of the pharyngeal parts, as well as superficial deposits upon the mucous membrane of the palatine tonsils. In other cases the first days may run a course without any symptoms pointing to the threatening danger. Besides the other symptoms which may be completely or irregularly developed, thus, the fever, the nervous disturbances, the cutaneous eruption, there is usually found a sharply circumscribed swelling and reddening of the mucous membrane of the palate and pharynx. With the full development of the eruption the fever begins to decline from its acme. A regular course appears to occur. The first sign that denotes disturbance is usually the condition of the temperature. Upon the morning of the fourth or fifth day of the disease the gradual drop in the temperature which has already begun is interrupted; either the morning remission or the evening exacerbation is greater than the day previously and from this time on the regular declining course of the fever ceases and the course becomes atypical. As the observation of this slight alteration in the temperature course is important, in prognosis as well as in treatment, I shall show some examples of this course in the temperature, with corresponding remarks. The underscored portions in the temperature curve indicate the change in the course of the disease.

The observation of the pharyngeal parts at this time does not always indicate the region in which the danger threatens, for which the temperature gives the signal of alarm; especially in little children, in whom the investigation of the pharyngeal areas is often difficult there is noted for the most part only a conspicuous increased formation of a thick tough mucus which



FIG. 29.—INITIAL RISE OF TEMPERATURE IN FOUR CASES OF SCARLATINA.

covers all parts, rendering an opinion regarding the nature of the beginning changes even more difficult. In older patients it may be noted how the coating upon the tonsils increases, or also in cases in which no superficial deposits are present it may be seen how a slight yellowish or grayish-yellow discoloration occurs in the tonsillar tumor, especially in its lateral aspect, particularly in the spaces between the palatine arches; or a hemorrhagic swelling of the

lower portion of the uvula, of the anterior surface of the palatine arch or similar conditions. Simultaneously with this a flow from the nose occurs, which does not, especially at first, produce anxiety. A thin, stale or even somewhat decomposed, yellowish-red fluid comes from one or both nasal openings and leads usually quite rapidly to an excoriation of the nasal openings or the upper lip. These excoriated areas show a lardy or dirty gray deposit. At the angle of the mouth small fissures appear (partly due to the forcible opening of the mouth, but even without this), which soon show a similar coating. Finally, there is added swelling of the lymph glands, first those of the angle of the lower jaw then also the neighboring groups, in the upper triangle of the throat, under the sterno-cleido-mastoid. These increase rapidly from day to day, from the size of a bean to the size of a hazelnut, or even attaining the size of a pigeon egg; they are painful to the touch. At first they are movable, soon, however, they appear in the underlying tissues, forming conglomerations of tumors.

Now the difficulty increases from day to day. The tongue becomes dry, the fever hovers around decided heights, the pharyngeal parts swell more markedly or—in the severest cases, those previously mentioned as resembling bubonic plague—in a brief period, in a night, they change into a leather-like, brownish-yellow dead tissue. In ordinary cases the discoloration primarily attacks circumscribed areas and rapidly produces tissue necrosis. After the end of the first week a part of the tonsil, the lower portion of the uvula or also a portion of the velum of the palate may be destroyed and be changed into an ulcer covered with necrotic masses. This is not only the case in visible parts but is not even infrequent upon the posterior surface of the velum of the palate. The diphtheroid has established itself about the choana, about the arch of the pharynx and is the cause of the ichorous flow from the nose. After existing for scarcely a week, as the result of these diphtheroid destructions, it may be noted sometimes even suddenly that a deep hole has formed in the anterior palatine arch, often circular or punched-out in appearance, in the depth of which the partly destroyed tonsil is found; at other times there is a deep fissure in the velum of the palate, which under some circumstances causes a fall of the entire palate upon the uvula, or upon the base of the tongue, at other times a separation of the uvula and the like. These destructions are always accompanied by a particularly profuse excretion of mucus from all the diphtheroid portions of the neighboring mucous glands. In some cases there is added a severe abscess inflammation of the entire oral mucous membrane accompanied by marked salivation; deep ulcers form upon the tongue, the lips swell enormously, are traversed by bloody fissures and disfigure the entire face.

Not infrequently does the diphtheroid inflammation attack the larynx, forming a rigid infiltrate of the ventricular bands and of the subglottal mucous membrane folds as well as numerous small discontinued desquamated deposits upon the surface of the mucous membrane. Both together then lead to stenosis of the larynx, which clinically is similar to true laryngeal diph-

theria, giving rise to the symptoms of croup and rendering tracheotomy necessary, which, in fact, only rarely avails to save the threatened life. If then the function of the velum of the palate is hindered, either by rigid swelling or by destruction, a condition resembling paralysis appears; there is nasal speech, regurgitation of fluid through the nose, symptoms which readily lead the inexperienced physician to the erroneous conclusion that he is dealing with true diphtheria.

Although the local signs may be well developed and distributed, they but very rarely cause the tragical termination of these cases. It rarely occurs that the destruction invades the tissue so deeply that a larger pharyngeal artery is eroded and a fatal hemorrhage occurs; this is an extraordinarily rare development. In the main, even these deep losses of substance heal with surprising rapidity if the organism is able to recover from the disease. The individual is particularly threatened by way of the glands and the blood vessels.

In the chapter upon pathological anatomy it was explicitly stated that this danger consists in the entrance of streptococci which produce inflammation and suppuration in the tissues and in the blood. There the various ways were described by which this occurs. The organism of the scarlet fever patient responds to this invasion in numerous regions by the appearance of hemorrhagic and purulent inflammations, the extent and number of which depend upon the nature of the entrance and possibly also upon the intensity of the original scarlatinal fever process.

A benign consequence of streptococcus infection consists in a more or less extended swelling of the lymphatics of the lower jaw and neck and the transition in one or the other gland into simple suppuration and abscess formation. Unilaterally or bilaterally upon the neck a circumscribed painful swelling appears, sometimes at the angle of the lower jaw, at other times lower down and more posteriorly, occasionally upon the sterno-cleido-mastoid, and in these cases it is usually combined with a transitory stiffness of the head and neck, and with a remittent febrile temperature. Without rupturing, the tumor may recede after existing for weeks, or very frequently in the first, in the second or even in the third week, a deep-seated and later more superficial fluctuation appears, the skin becomes red, tumefies and ruptures in case it has not been incised previously, and pus is discharged.

Frequently suppuration does not occur but dry necrosis takes place. One gland after another enlarges, with continued high temperature, until tumors the size of a walnut and larger develop; the surrounding connective tissue becomes tough, and in the entire region there is a board-like infiltration, individual areas of the skin are discolored, becoming dark blue and then showing gangrenous decomposition; if these parts are incised with the hope of finding pus, a dry, almost cheesy tissue of a grayish-red color is met with that shows the distinct characteristics of gangrenous substance. It is fortunate if by means of a profuse suppuration in the surrounding tissues these dead masses are desquamated and discharged. Much more commonly even these show a gangrenous decomposition and upon both sides of the neck after prolonged



fever, markedly distributed, deep losses of substance are noted, the carotid being seen to pulsate at the base, and in some instances even the wall of this vessel has become eroded and a fatal hemorrhage has terminated life; however, death usually takes place without this complication, due to the exhausting fever and the septic consumption. Frequently the local diphtheroid process takes place in the ears as well as in the nose and in the conjunctiva and before death relieves the child both eyes may be totally destroyed due to the inflammatory necrosis.

Again, in other cases the infection runs its course without such conspicuous symptoms but is no less threatening. Slowly the purulent infiltration creeps along the trachea, in the glands, and the neighboring connective tissue, invading deeply and, finding entrance into the thoracic space, it leads to purulent mediastinitis, to which often weeks later a large ichorous empyema or a pericarditis is added. To what tortuous tracts this latter fatal affection may lead I have observed twice, in cases in which a purulent meningitis caused the fatal termination, that this had not originated from the nose or the ears but was primarily of a spinal nature, thence having ascended to the brain. In the previously described manner a posterior mediastinitis had taken place, from which a purulent right-sided pleuritis had originated, and thence the suppuration passed through the intercostal nerves, and traversed the intervertebral foramen, thus reaching the spinal cord cavity.

Probably some general purulent cases of peritonitis which are met with in connection with scarlet fever develop in the manner indicated here, by continuity. But just here it may be mentioned that occasionally also *appendicitis* may precede. I have noted the appearance of this disease a few times in the course of scarlatina; a similar relation of conditions has lately been noted between purulent typhlitis (*appendicitis*) with purulent inflammations of the tonsils, the same genetic connection existing.

If these serious changes which have just been described regarding purulent infection remain local and its distribution is limited to the neighboring lymph glands and lymph spaces in the connective tissue, in another series of cases it arises by means of metastasis in areas quite removed from one another, provided streptococci in larger numbers find their way into the blood. The great number of these organisms in the blood can always be shown by bacteriologic investigation. In connection with Bahr<sup>dt</sup> I published the first case of this kind in which the mere microscopic examination of the blood and of the products of the purulent metastasis showed the general proliferation of the microbes through the entire organism. The way in which this rupture occurs has been directly shown in individual cases to be due to an original diphtheroid affection of the pharyngeal passages, a septic, inflamed and thrombosed vein being near by in which a septic decomposition of the clogged coagula has occurred. Here by means of continued, usually high, fever, in which, however, chills occur in all possible parts of the body, in the subcutaneous tissue, in the muscles, the kidneys and in other internal organs, smaller and larger abscesses develop: a true pyemia. The joints and serous membranes, espe-

cially the pericardium, are by far most frequently affected by this scarlatinal pyemia. The joints in large numbers, the small as well as the large joints, are then subject to purulent infection. The knee joints, the elbow joints, the wrist joints, on one or both sides, especially also frequently the finger joints, enlarge so that there is marked pain upon touch, the patients who are usually almost comatose in this condition manifest the pain by loud cries. The cutaneous covering is reddened in a wide area above and below, swelling in a manner sometimes resembling erysipelas. It is terrible to see how such joints are completely destroyed in a few days, so that upon careful examination with careful movement, the completely destroyed cartilage is felt detached from the bared ends of the bones.

But even with these catastrophes the list of lesions which may arise from scarlatinal diphtheroid is not yet exhausted. The distribution of the local mucous membrane disease has not yet been mentioned or but briefly indicated, which still contains a quiver full of dangerous arrows: *disease of the ear*. This is a very frequent complication of scarlatina. In the hospital I noted it in 27.4 per cent. of all admitted cases (393 patients in the years 1894 to 1897). In the usual manner, by way of the Eustachian tube, the severe mucous membrane affection maintains its character in the pharyngeal cavity, showing a tendency to inflammatory necrosis of the mucous membrane and neighboring bones. The milder forms of otitis media lead to simple perforation of the tympanic membrane, whether this occurs naturally or artificially, terminating in slow recovery with profuse suppuration. Very often, however, this does not terminate the condition; in spite of sufficient opportunity for the pus to flow

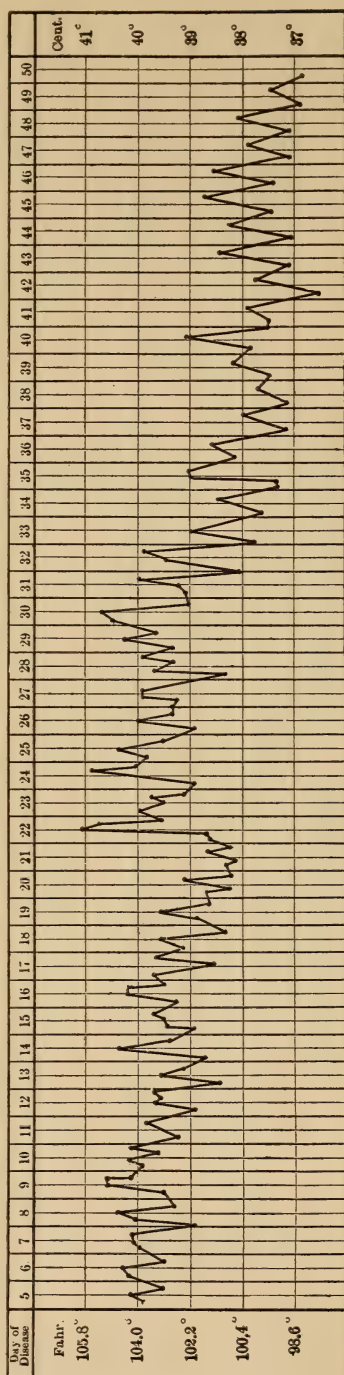


FIG. 30.—TEMPERATURE CHART IN COMPLICATED SCARLATINA.

off, the inflammatory necrotic affection continues into the antrum and to the cells of the mastoid process, in which the osseous tissue itself is implicated and changed into an ichorous necrotic inflammation. From here the infectious process finds its way to the sinus or to the meninges. And in this manner either a general blood affection, a pyemia, is produced, or, by absorption of the poison from the ear, a fatal septemia or a purulent meningitis leads to the lethal termination.

The scarlatinal otitis just described, in its severer forms, is always accompanied by marked and tenacious fever, which even with proper treatment may last for weeks and lead to a severe consumption of bodily strength, so that then only slightly increased disturbance is sufficient to entirely obliterate the remaining feeble strength. Persistent unrest at night, insomnia, delirium, usually a high grade of anorexia, diarrhea and bronchitis belong to the complications which increase the ear difficulty.

The temperature curve (Fig. 30) on page 693 shows a picture of the long-continued condition occurring in the child as the result of diphtheroid and severe otitis, until finally recovery took place.

In this case the appearance of the exanthem was delayed, appearing upon the fourth day and being but little characterized; only upon the eleventh day was there marked swelling and redness of the tonsils with a deposit upon their surface. With a rise of temperature, upon the fourteenth day of the disease the otitis media became prominent. Upon the sixteenth day a high grade of edema was present in the region of the right mastoid process, and in operation upon the seventeenth day the bone was found discolored to a great extent and necrotic, the antrum being filled with granulations and discharging ichorous pus. Slowly and gradually the desquamation of the necrotic osseous tissue took place.

Upon the twenty-second day of the disease there was a sudden exacerbation of the condition with a severe chill. Now the sinus transversus was exposed; puncture, however, did not show the presence of pus. The internal jugular vein was tied with a hook upon the right side to prevent the entrance of septic products from the ichorous ear.

In spite of the continuation of the irregular septic fever until the fifth week of the disease, gradual improvement occurred in the osseous wound. Finally, complete recovery was still further interrupted by a spasmodic attack upon the forty-second and forty-fifth days of the disease which, however, left no deleterious consequences. Ultimately hearing in the right ear returned and was quite good.

At the end of the twelfth week the little patient visited me in my office. The wound in the ear had healed.

In cases of this kind the help of a skillful ear specialist is required, as delay and temporizing is not in place in these severe forms of scarlatinal otitis, in which every day may bring about a serious condition, but the diagnosis must be followed in these cases by an immediate life-saving operation. It is a mistake to dread "interference with the natural process of healing," for this is as good as impossible or represents but an exceptionally fortunate termination. Usually the case runs on as in the following instance when an operation was attempted:

Boy aged thirteen and one half years was taken ill upon December 13th, with a medium severe attack of scarlet fever. Defervescence occurred about December 20th,



and upon December 21st there was an exacerbation and the development of otitis media. In spite of the appearance of the tympanic membrane and the auditory canal denoting trouble, the posterior wall swelling out and even granulation formations occurring, neither the family physician nor the specialist who was called in consultation could conclude to operate upon the mastoid process. In the beginning of January the fever became higher, strength declined more and more and now another ear specialist was called in, who immediately opened the mastoid process and found the entire bone completely softened, and all of its cells filled with pus. Following operation, there was general improvement and decline of fever. But even upon January 3d, there was severe pain in the splenic region, followed by a septic peritonitis, jaundice, and death upon January 5th.

Probably even before the final opening of the pyogenic focus, sinus thrombosis and septic splenic infarct had occurred.

But even without such fulminant phenomena having preceded, we are often surprised by the consequences of an insidious and treacherous disease of the tympanic cavity and of the antrum.

I recently saw an instance of this kind. Upon July 2d, a girl aged twelve years was attacked by scarlet fever, which appeared to run a mild course, so that only on account of the great anxiety of the parents regular visits were continued up to the third week. Upon July 19th, an otitis media began to develop, paracentesis being performed a few days later. Upon July 25th, the mastoid process was moderately painful, but as no fever had developed it was, nevertheless, advised to open the bone. The operation took place upon July 27th, and great quantities of thick yellow pus were discharged. Upon July 28th, pain in the back, and in the course of the day headache, eleven o'clock in the evening temperature of 104° F., pulse 160. During the next few hours great unrest, rigidity of the muscles of the neck; death upon July 29th, at a quarter past one o'clock in the morning.

In this instance, in a very rapid manner and in an originally very mild case of inflammation of the middle ear, a fulminant meningitis occurred.

In several instances in which there was an otitis media with an apparently good flow of pus, a continued fever was present which could not be explained and in which it was advised to open the mastoid process, in spite of the fact that no swelling of its coverings could be determined and only slight pain upon pressure and the swelling of a lymph gland lying behind the ear pointed to its implication. Invariably, pus and granulations were found in the cells. The fever disappeared and recovery occurred. In cases of this kind, naturally, the opinion of a specialist who has been called in consultation must be considered and he must be supported if he decides in favor of an operation. Instances like those just described rather incline us to take upon ourselves the reproach of polypragmaty than to be guilty of omitting an operation which perhaps might save life.

**Scarlatinal Rheumatism.**—Much less serious are those irregularities in the course of scarlatina, due to swelling of the joints, which is usually designated by the name of *scarlatinal rheumatism*. This must not be confounded with the previously-mentioned joint affection which almost always is of an unfavorable prognosis.

Scarlatinal rheumatism is not a very frequent complication. I noted the

condition 29 times in 358 cases, therefore, in 8 per cent. In my Children's Hospital it was found in 6.7 per cent. of the cases. This proportion allows of the conclusion that the affection is not in immediate connection with the scarlatinal poison but is due to special auxiliary causes. The pathogenesis is not accurately understood, only this much is certain that this multiple joint affection occurs in no other infectious disease as frequently as in scarlatina (acute articular rheumatism naturally excepted).

Clinically, the affection resembles acute articular rheumatism in many respects, but in the main it is more transient in character and of briefer duration than acute polyarthrititis.

In rare cases the condition may be noted upon the first day of the disease; not only as a general muscular pain, but with distinct localization, for example, in the feet. As a rule, it occurs during the second half of the first or the first half of the second week, therefore, in the period between the fifth and twelfth days of the disease. The large as well as the small joints of the extremities are affected. The vertebra, the jaw, etc., I have never seen implicated. Scarlatinal rheumatism appears to have a special preference for the wrist joints, and also the finger joints are not infrequently attacked. But the legs are by no means spared, the knee joint, the ankle joint, sometimes the joints of the toes and even the hip joints are attacked. Occasionally the affection is only limited to a pair of joints, for example, both knee joints, both wrist joints. Sometimes the affection wanders from one place to another, similar to the condition in primary articular rheumatism.

The local symptoms are quite similar to those in acute rheumatic fever. Occasionally there is no swelling or redness in the region of the joints, and pain upon pressure and pain upon spontaneous movements—but this may occur with great severity—show the implication of the joints, sometimes, however, there is marked periarticular swelling and redness or a distinct effusion into the joint may be noted. Occasionally I found that the entire skin of the feet or the hands was edematous, and once I observed, during an unusually prolonged attack of scarlatinal rheumatism lasting ten days, the appearance of a general (vaso-motor?) anasarca without any signs of a nephritis. The cutaneous edema disappeared simultaneously with the rheumatism.

The duration of the complication is almost always brief, limited to a few days (three to five). In how far the antirheumatic treatment which was employed by me may have caused this cannot be determined with certainty. Some few cases that I saw during the early periods of my practice, before the action of salicylic acid was discovered, ran a similar brief course to those that were treated.

A remarkable fact is that the *heart* is implicated in a like manner to that occurring in acute articular rheumatism, regardless of whether arthritic pains appear or not.

As a rule, these *endocarditic processes* are of a *benign* nature. They must be entirely separated from malignant affections occurring by way of

metastasis (similar to the analogous joint affection), particularly from those due to the diphtheroid affection of the pharyngeal organs and cervical lymphatics which preferably attacks the pericardium, but may also implicate the endocardium, causing a rapid destruction of the valves of the heart and leading to septic infarcts in various internal organs. Another form of cardiac disease which was previously mentioned in the description of scarlatina gravissima and which we shall mention again in the description of scarlatinal nephritis, consists in an intense (toxic) damage of the *heart muscle*, the myocardium, showing itself by a dilated cardiac weakness, occasionally in the form of sudden cardiac death. From these affections also the rheumatic, or rather rheumatoid, form of scarlatinal endocarditis, which we must consider now is to be entirely separated. It develops occasionally in the same period with the rheumatoid joint affection, therefore, between the first and second weeks of the disease. Sometimes the signs of the affection are only noted after complete disappearance of the eruption, improvement of the scarlatinal affection and the return of subjective well being, during an apparent period of convalescence. Subjective symptoms are almost entirely absent and the complications cannot be discovered without auscultation; by this means, however, most markedly at or near the apex, but sometimes even most plainly at the base, a brief or longer soft murmur, synchronous with the ventricular contraction will be noted. A little later perhaps also a moderate distribution of cardiac dulness and a slight accentuation of the second pulmonary sound may be demonstrated. The patient who is still in bed does not complain and perhaps only a somewhat marked pallor of the face is conspicuous. Sometimes endocarditis is combined with pericarditis, and then with the corresponding objective phenomena decided symptoms appear.

It will be noted in not a few such cases that these signs disappear entirely, and in those cases in which there is an opportunity for prolonged continued observation the patients recover completely without signs relating to the heart. To a certain extent it always remains questionable whether there is an endocarditic process or whether only so-called accidental (functional) murmurs are present. But, on the other hand, from these rheumatoid, scarlatinal endocardites, unquestionably, valve lesions arise. Not infrequently is there an opportunity to note this, as the cases of cardiac murmurs, which are not very rare, that have positively developed during the course of scarlatina, are almost always found to disappear. I am, however, in possession of the facts of a case in which from a previously intact heart during an attack of scarlatinal rheumatism, the appearance of a murmur was observed and the gradual development of a typical mitral insufficiency could be followed step by step. Thus the scarlatinal infection in this direction is also treacherous, not being immediately dangerous to life but permanently damaging health. That in scarlatinal endocarditis the musculature of the heart (or the nerve-cells of the heart?) is not spared may be concluded from this, that during the existence of a cardiac murmur very frequently a more or less great irregularity of the pulse is observed. This is by no means so rare, even though there be no opportunity



of demonstrating an endocardial complication occurring particularly in the stage of convalescence, often only in the sixth or seventh week and even later and then disappearing. This appears to occur particularly in those cases in which the affection is prolonged by nephritis.

The so-called *scarlatinal typhoid*, the relations of which to the disease are not yet quite clear, but which for the most part represents a benign and by no means frequent deviation from the normal course: A fever accompanied by moderate symptoms of depression and occasionally of marked disturbances on the part of the digestive organs and lasting for a long time without demonstrable local disturbances. Even this conception shows that not every febrile sequel of scarlatina that cannot be easily explained can be designated by the above name. And in every fever of this kind it is our first duty to search for the point at which resorption of material which may produce fever is possible. Such areas are numerous enough in the case of scarlatina. In my experience, especially the posterior pharyngeal wall and the posterior nares as well as the nasopharyngeal space should be considered. If there be seen—even although a previously present pharyngeal diphtheroid has disappeared—upon the posterior pharyngeal wall any decided muco-purulent secretion, a roughening, a superficial erosion or granulation of the mucous membrane, the possibility that the fever is due to this cause must not be overlooked and the result of a suitable local treatment often enough shows the correctness of this assumption.

The second point of origin of the septic post-scarlatinal fever is in the lymph glands. If these are found hard upon one side or both sides and large and sensitive, the fever may be due to them even if they do not always suppurate. A third point of origin may be traced to one or the other region of the middle ear, which has already been mentioned above.

But, besides these septic post-scarlatinal fevers, there are individual cases in which even the most exact and careful investigation will not reveal the source of the fever and in which the temperature curve does not at all show the intermittent or remittent character of resorption fever, but shows moreover a certain similarity to typhoid fever. As an example, the following temperature chart in which this fever occurred between the eleventh and twenty-third days of the disease, is shown (Fig. 31).

A boy aged two and a half years was attacked, upon April 19th, with vomiting and fever, simultaneously with hoarseness and cough, in the midst of complete health; upon the second day of the disease, upon the arms and legs as well as upon the buttocks, there was at first a non-characteristic, later, a decided, eruption with marked redness of the pharyngeal areas. Upon the seventh day of the disease desquamation occurred, which continued into the fourth week, the scales consisting of large and long lamellæ of the epidermis, from the gluteal region as well as from the ribs. The cutaneous redness as well as the angina had disappeared in the second week, only at the angles of the mouth fissures formed with a lardaceous coating which required a long time for healing. No lymphatic enlargement or otitis developed. The kidneys remained intact. For the continued fever from the tenth day on there was no explanation. A dry bronchitis occurred, which continued for a long time, the abdomen from the fourteenth day of the disease became tympanitic, was painful to pressure, and diarrheic stools of a greenish-

yellow color appeared. The spleen was not palpable. The child was irritable, now and then vomiting recurring, and with these symptoms the affection continued even longer than the time shown in the temperature curve. After the twenty-third day of the disease the temperature was no longer regularly taken; toward the middle of May recovery gradually occurred.

Apparently the process which produced the fever in this case developed in the digestive organs. It has been previously mentioned that under the influence of a scarlatinal affection, the mucous membrane not only of the nasopharyngeal space, but under some circumstances that of the entire intestinal tract shows hyperplastic swelling. It might be supposed that in such cases resembling a mild enteric course this deleterious influence of the scarlatinal poison affects the adenoid tissue which is so profusely present in the intestinal tract, as well as the mucous membrane, and thus leads to a condition of the

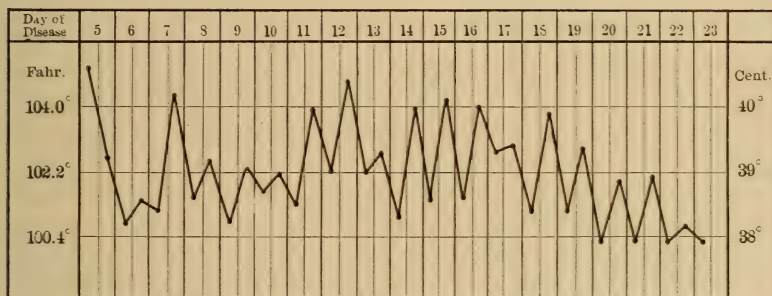


FIG. 31.—TEMPERATURE CHART OF SCARLATINAL NEPHRITIS.

digestive organs which resembles that brought about by the pathogenic agent of enteric fever. This cannot be determined with certainty as cases of this kind are not subject to anatomical investigation. In concrete cases, one point is still to be observed. Occasionally at the onset of enteric fever an eruption occurs which may closely resemble the exanthem of scarlatina. In such genuine cases of enteric fever with pseudoscarlatina, the appearance of the rash (roseola), the proof of typhoid bacilli and agglutination are the points of support for the correct diagnosis.

A rare deviation of the regular course of the disease is shown by the *relapse*. By this we do not understand an increase of the redness upon the skin, which is frequently observed, causing the eruption to become plainer, which shows itself by a new rise in temperature, but the return of all of the symptoms of scarlatina, or at least of the eruption, after a complete cure of the primary affection.

This relapse which resembles the condition arising in enteric fever occurs in individual cases.

A boy aged seven, who suffered from a mild attack of scarlet fever from June 24th to July 8th, after he had been out of bed showed upon July 19th a decided swelling of the cervical lymph glands; following this, upon July 21st, with moderate fever, a

new exanthem occurred, which in individual areas consisted of large macula and in others of small macula, having partly a measly appearance. Upon July 25th, the fever disappeared, swelling of the glands declined, and the eruption could no longer be noted.

In another instance a boy aged four was attacked upon June 30th with scarlatina, with quite an intense diphtheroid and lymphadenitis. During the period of desquamation, in which the angina improved, upon July 13th, with renewed but brief fever, a fresh eruption occurred upon the right forearm and right thigh.

In the course of a gland abscess during the existence of a nephritis I saw upon the fourteenth, and also upon the twenty-third, day of the disease a relapse and appearance of the exanthem with high fever. These relapses do not appear to be serious.

If the patient has resisted or escaped all of the described dangers which may occur in the course of the first two weeks of scarlatina, he has not yet escaped all of the dangers of one of the most treacherous infections; for, unexpectedly, after he appears to have been on the high road to recovery, often without any warning, an *affection of the kidneys* may attack him, which we meet with in many other affections, but in no other does it occur so frequently or so severely or take on such a substantive character as in the case of scarlatina. *Scarlatinal nephritis* may be looked upon as the prototype of infectious nephritis.

**Scarlatinal Nephritis.**—It is by no means a necessary consequence of the infection, as only a part of those affected, and at that a small part, shows this complication. In fifteen years in my private practice I observed 36 cases of scarlatinal nephritis in 358 cases of scarlatina, therefore, almost exactly 10 per cent. In 393 cases in the course of four years, in the Charité in Berlin, 77 cases showed a nephritis, that is about 20 per cent. (19.6 per cent.). This difference, however, is not so great as it appears, as in private practice there are many more mild cases than are met with in the hospitals. The epidemics of individual years vary greatly, climate and perhaps racial characteristics appear to play a certain etiological rôle. Johannessen reports of some Norwegian observer that he had 90 per cent. of renal affections in scarlet fever patients. But even in Norway the statistics of those physicians that have had a large experience do not rise above 20 per cent.

This is a remarkable difference from those complications which develop from the point of entrance of the supposed poison in the pharyngeal organs. Here in 60 per cent. of the hospital patients we found a diphtheroid mucous membrane inflammation.

What condition of the total disease brings about the renal inflammation is still unknown. Evidently it cannot be sought for in the severity of the infection at the onset, if we may judge from the intensity of the symptoms. For in quite a number of cases of nephritis, among them particularly the severest (for example, in my practice of 5 fatal cases, 4!), a mild, sometimes scarcely noticeable preliminary affection, with slight fever, fully developed eruption and mild angina precedes the complication.

It has already been mentioned above, that particularly the rudimentary



cases often only become dangerous from the renal complication. And the period of the appearance of the renal affection in the course of scarlatina is against the view that it is an immediate action of the contagion. On the other hand, unquestionably, the development of scarlatinal nephritis from accidental external influences, possibly refrigeration, leaving bed too early, errors in diet and the like, may be rejected, for the development of nephritis cannot be prevented by the most careful nursing and diet and it affects those that are kept in bed constantly and patients that are only nourished with milk,<sup>1</sup> to the same extent as those not so carefully treated. The influence of errors in régime are not to be entirely denied, but these must only be looked upon as auxiliary causes and do not by any means play the chief part, as, for example, in the development of pulmonary complications in measles. Especially the so-frequent appearance at a distinct period of the course stamps nephritis as an affection belonging to the scarlatina process. It is a late action of the infection. That it occurs at all is perhaps favored by a special sensitiveness of the organs or loss of resistance. The occurrence of such relationships of individual cell groups to certain poisons, particularly also to the group of parasite toxins, has been sufficiently determined with certainty by the new experimental etiology. Clinically, this view is favored by the circumstance that not so very rarely a distinct family predisposition to scarlatinal nephritis is met with in so far as often two or more children (I myself saw four) of the same family simultaneously show nephritis after scarlatina. It can scarcely be doubted that the kidney forms one of the excretory organs of the abnormal products, the symptoms appearing after the infection and in this manner the "haptophorous" cell groups of the organ may become diseased.

A fact which at first glance is peculiar has been mentioned several times, that this renal inflammation occurs during the period in which the rest of the process, the throat affection, the rash and the possible joint implication have disappeared. The first period of the symptoms which denote a renal affection appears at the end of the second or in the third week. Among my 36 cases, which for a great part I observed from the onset of the disease, the affection began seventeen times between the twelfth and fifteenth, ten times between the seventeenth and nineteenth days of the entire process. In the other cases the period was uncertain, or the onset of the affection occurred in a later period. Exceptionally, the renal disease may even occur several weeks after an intervening period has preceded. Lately I noted this complication in the thirty-eighth day of the affection in a girl aged two years and nine months.<sup>2</sup>

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<sup>1</sup> That is not always an advantage. Exclusive milk diet is apt to cause indigestion, indicanuria, and subsequent renal irritation. It is about time we should change that exclusive milk diet into a mixed milk and cereal diet. It takes a long time before the results of biochemical studies are utilized in practice.—EDITOR.

<sup>2</sup> In my experience nephritis may be looked for on the ninth or tenth day, and onward, up to the twenty-third, after which it is rarely observed. I have seen it to appear on the fifty-fourth day of a scarlatina in a girl of seven years exhibiting a very slow desquamation with no other bad symptoms.—EDITOR.

The child was taken ill on January 1st, and upon January 5th was sent to the Charité. The course of the disease was not particularly severe, being combined with a mild diphtheroid. The urine, constantly examined, was still free from albumin upon February 4th. Upon February 7th, a mild hemorrhagic nephritis occurred, which began to disappear upon March 3d.

This paradoxical condition, which is analogous to certain symptoms of late diphtheria, as well as to some infectious diseases of very long incubation, was explained by some physicians in that they assumed that the renal affection existed from the onset of the disease and only produced symptoms late in the course of the malady. It is true that with high fever, in the first week albuminuria and casts occur in the urine; but after defervescence this disappears completely. In dozens of cases which were carefully examined for this sign, various authors, especially Thomas, found that the composition of the urine for days and weeks after the primary febrile albuminuria was again completely normal before scarlatinal nephritis quite suddenly appeared. From my own experience, I may throughout confirm the views of Thomas. According to my investigations, as well as those of other authors, for example Sørensen, the anatomical changes in the kidneys of scarlet fever patients that perish in the first week are entirely absent or but very insignificant. It is not easy to comprehend how especially the last named author refers the development of nephritis to the first period of the entire process. This view nowadays is shared by but very few physicians.

In a pathologico-anatomical, as well as in a clinical respect, scarlatinal nephritis shows a character which naturally is also peculiar to other infectious renal inflammations, but which is particularly prominent in scarlatina, and at least in the recent cases and in those that are at all well developed it is never absent: This is the hemorrhagic character of the pathological process. In the anatomical examination this is well defined to the careful observer, even if not always macroscopically still microscopically, naturally, provided that the organ is not hardened in alcohol, which destroys the hemoglobin. Sørensen found in 15 carefully examined cases of scarlatinal nephritis numerous, mostly hemorrhagic casts in the uriniferous tubules, and in 11 cases several times, "yellowish masses" in the capsular space. Kaufmann describes acute hemorrhagic change in the kidney due to scarlatina. I have examined various cases of scarlatina carefully and agree with those observers that have found at the onset of the disease, especially in the grave cases that succumb early, a marked hyperemia of the organ but as yet no signs of inflammatory changes. Only during the period in which the children succumb to the consequences of nephritis (cardiac asthenia and dropsy, especially pulmonary edema or uremia) does the kidney show distinct signs of hemorrhagic inflammation, above all, in the glomeruli. The accompanying figure (technically a not very complete reproduction of a photogram) gives an example of such a hemorrhagic glomerulitis.

The thick opaque capillary loops, rich in nuclei are seen, from which intense hemorrhage (*b*) has occurred. These fill a part of the capsular space,

the epithelium of which has proliferated. The convoluted uriniferous tubules near the glomeruli from which hemorrhage has occurred are filled with the exuded blood (*cc*). But we are not dealing here with a simple collection of red (and corresponding white) blood corpuscles in the lumen of the canal, but its entire contents, usually including the epithelium, have been moulded together in a coagulated mass, in which no longer any nuclei of the epithelia are discernible but only a homogeneous or slightly granular mass is noted, in which red blood corpuscles, usually of a small diameter, may be seen. Here, therefore, the blood which has exuded from the glomeruli as it appeared, produced a toxic action of the epithelia. In such areas, by means of methods which allow the fact to appear, always larger or smaller globules of fat partly still in the form of epithelium may be noted. In the loops of the uriniferous tubules I have never seen such hemorrhagic coaguli. Hemorrhages, however—in so far as we are justified in concluding from position and form of the epithelium—occur also in the spaces between the

renal tubules; here, however, they are not combined with such epithelial necroses but simply lie in the lumen, which could occur in no other manner but by diapedesis. In the collective tubules, finally, clogging by hyaline casts is found and here also without deeper implication of the epithelium, the nucleus of which is still retained. Only here and there a desquamation of the entire epithelial cylinder from the basic membrane of the tubule is shown. The massive clogging of the straight canals (here and there also of the loop canals) has a further consequence: that of urinary stasis in the entire area of the affected collective tubules. Thus the convoluted canals of the labyrinth which are not affected in the previously described manner are found *dilated* to a great extent, their lumen large, their epithelium (otherwise unaltered) pressed against the wall. This is also true of the intercalary portion as well as of the

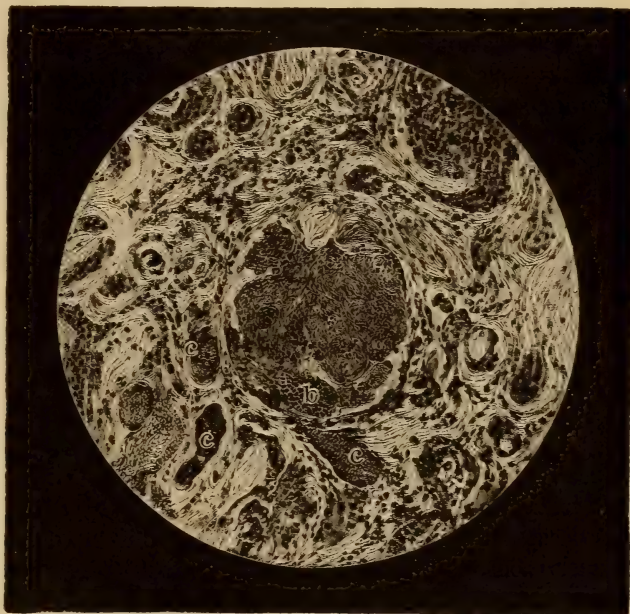


FIG. 32.—MICROSCOPIC SECTION OF KIDNEY IN SCARLATINAL NEPHRITIS.



convoluted tubules. The damage of the active part of the kidney consists, therefore, in a disturbance of the normal function of the glomeruli (especially in the excretion of water), secondly in a destruction of a part of the secreting epithelium of the labyrinthine part of the canals, and thirdly in stasis of the secretion in the remaining part. This is the process in the pure, severe cases (for example in a case dying after nine days of anuria).

In the milder cases, the two deleterious effects upon the parenchyma of the kidney are of but slight extent, whereas the hemorrhages from the glomeruli may even be considerable. In the formation of casts in the uriniferous tubules, it will depend particularly upon their density and adherence as to whether the consequences will be severe or mild.

From the observation of these findings (fresh frozen sections of kidney hardened in formalin) it will be seen that in this form of nephritis the *primary* damage occurs in the *vascular* arborization, therefore, in the interstitial part of the glandular organ (this, for example, is not so marked in the diphtheritic kidney). Most noticeable in the glomeruli, and first studied by Klebs and afterward by numerous other investigators, this pathological change has led to the designation of the scarlatinal kidney as a glomerular nephritis. But this does not mean that only the glomeruli are implicated. Although I cannot recognize the periarteritis described by Fischl in the small arteries, the circumstance that the capillary net which surrounds the labyrinthine tubules allows blood to exude proves that even there the endothelial covering of the capillary tube is diseased. This may be more marked in the secretive portions of the capillary system than in the nutritive area.

That in consequence of the damage to the capillary wall, not only the previously described hemorrhages but also in more or less numerous areas, collections of leukocytes appear around the vessels, is readily comprehended. In the *recent* cases of pure scarlatinal nephritis, this finding plays no great rôle. Particularly dense is this noted in areas in the boundary layer in the vicinity of the curved arterial twigs, around the glomeruli and upon the surface of the kidney.

If the scarlatinal nephritis has lasted for weeks or months previous to the anatomical examination, the interstitial conglomerations of cells may reach high grades and may lead to an extended change in the parenchyma, necrotic areas, connective tissue proliferation and contraction, producing an entirely different picture. But even in these cases the interstitial inflammation will be prominent. *Septic renal disease* which occurs in connection with severe diphtheroid, in a restricted sense differs in its anatomical appearance from the scarlatinal, more closely resembling other septic processes.

In fatal cases in which scarlatinal nephritis enters upon a chronic condition, it usually takes the course of a very slow contracted kidney, which requires at least a few years for its development.

The comparatively slight mortality from scarlatinal nephritis, in spite of the frequent threatening symptoms, denotes in the main that the damage to the kidney is not too severe. Of 36 cases observed by me, 5 died, a mortality

of 14 per cent. In the hospital, during the years 1894 to 1898 the mortality from renal inflammation amounted to 26 per cent.

Clinically, the appearance of nephritis in the scarlatinal picture in quite a number of cases is not characterized by subjective symptoms. The parents notice a disturbance of convalescence in their child, either by a swelling of its face or by the odor or composition of the urine. More than once have I been called by a mother to see her child, with the remark that its urine resembled hers during the menstrual period. Otherwise the child was quite well. With these symptoms the inflammation of the kidney not rarely runs its course from beginning to end.

In other cases the disease begins with new general symptoms. The temperature rises, either in the form of a single daily rise to a moderate or even to a decided height (over  $104^{\circ}$  F. even to  $105.5^{\circ}$  F.), with chills, burning or sweating, or the fever continues for several days, even lasting for weeks, showing a remittent course. The cause of these variations in the reaction is not known. The intensity of the nephritis and of the fever do not always run parallel.

Then vomiting is often frequent, which may be repeated many times during the first days, to this there is added headache and restless sleep; anorexia is also present.

The pulse often rises with the fever, but, on the other hand, abnormal slowing and arrhythmia are observed. Thus in a boy aged seven years, upon the sixth day of a febrile nephritis (terminating in recovery), I counted 64 regular beats per minute, in another case, in a boy aged eleven years, a nephritis began upon the nineteenth day of the scarlatina, the pulse was from 96 to 100 per minute; in the second week of the renal inflammation it fell to 60 to 66, rising again as recovery advanced.

Slight swelling due to dropsy of the subcutaneous cellular tissue can always be observed upon careful examination; above all, a slight edema of the face and a brief pitting upon pressure with the finger upon the tibial surface and upon the sternum. Often dropsy is much more marked, especially in the legs and upon the buttocks, being combined with a watery transudation in the abdominal cavity, in the pleura and in the pericardium.

If the urine has not been examined previously the symptoms just mentioned lead the physician to an examination and now the great change that has occurred is discovered. In place of a light yellow or light orange colored, clear, profuse urine, its characteristics a few days previously, a turbid reddish-brown or dark red, opaque fluid is noted, which shortly after standing deposits a more or less dark cloudy sediment. If the daily quantity is measured it is found to be decreased, for example, from the previous 1,000 to 1,200 c.c. its volume may be decreased to 600, 500, or even less. In keeping with this, the specific gravity is raised. An examination of the filtered fluid shows more or less marked contents of albumin (2, 3, up to 7 per thousand and more) and the dark color is shown to be due to an admixture of blood, the coloring matter being determined by the guaiac test or by Heller's sediment

test. If the admixture of blood is not very marked, the urine is light red in color, sometimes resembling the washings from beef. If held to the light it is shown to have a greenish tinge.

An examination of the sediment shows numerous erythrocytes; they are often conspicuously small and not, infrequently, besides a small undamaged erythrocyte, or an erythrocyte appearing as if it had been dissolved, a brown granular detritus may be noted even in freshly voided urine. From this it must be concluded that not rarely also in the kidney, perhaps even in the renal vessels, a decomposition of the red blood cells has occurred. I noted once during the period in which nephritis usually occurs a severe hemoglobinuria with a rapidly following fatal termination.

Besides blood corpuscles, there are found in the sediment various hyaline casts of varying character, large and small, long and short, smooth and uneven; often red blood corpuscles are found enclosed in the casts or are adherent to them. Some casts are purely epithelial; some are impregnated by urate salts, others carry finer or coarser globules of fat, again others contain leukocytes. Finally, waxy casts are also seen. In recent cases, leukocytes are not very predominant, renal epithelium is not scant. Cells with fat granules are quite rare; I once saw them on the first day of a nephritis in a boy aged seven years; the course of the affection was mild.

Besides these changes, which are due to the anatomical disturbance in the kidney, in the course of the last few years it has been attempted to determine the diminution in functional activity somewhat more accurately, in that the effort has been made to determine the change of the molecular concentration by means of physical examination. The results obtained by *cryoscopy* of the urine up till now are, however, by no means conclusive. The function which can be determined by simple measurement, for the present, is the only practical one from which we may form an opinion, both in a diagnostic as well as in a prognostic respect: *the excretion of water*. The lower this falls the more serious the prognosis. The daily amount of urine, not the albumin contents, and certainly not the composition of the sediment, inform us regarding threatening danger.

In the mild cases the excretion of urine does not fall below 400 to 600 c.c. per day. For a week or two the previously described composition of the urine remains the same, the daily quantities become larger, the color lighter, and the daily quantity of albumin smaller. Finally, the albumin disappears entirely. But the sediment does not disappear at once; even days after no trace of albumin can be proven by the most delicate tests, the desquamation of epithelium and casts still continues. The total duration of the affection is about three weeks. The subjective difficulties are always moderate; a certain lassitude is even shown by the patient that remains in bed and he always shows an unusual pallor and swelling of the face, even in the cases in which no marked dropsy is present. In fact dropsy may be absent entirely in the mildest cases. Gradually as recovery occurs the normal healthy color of the face returns.

This is different, however, in severe cases. The subjective difficulties are



greater and very often the patient complains of pains in the renal region which are increased by pressure; these pains are otherwise generally localized to the abdomen. I have never seen severe attacks of colic due to acute inflammatory changes in the kidney as have been described by Israel.<sup>1</sup> The appetite is gone and diarrhea is often present. The pallor is of a high grade, the face occasionally appearing waxy pale, almost transparent.

The diminution in the amount of urine is conspicuous; in a few days the excreted amount falls to 150, 100, 50 or even 0 c.c. and this anuria may last for days (in a case recently observed nine full days passed before death occurred). However, in the urine that is excreted there is sometimes observed the paradoxical phenomenon that it contains less of a pathological admixture regarding albumin as well as sediment than the previous profuse urine. This can hardly be explained in any other manner than that such a secretion occurs from areas of the kidney that are but slightly attacked, the convoluted tubules of which are not clogged. The anatomical investigation in the severest cases also shows by no means a simultaneous severe affection in all areas of the glandular organ.

Now, as a rule, but a few days pass before the symptoms of *uremia* appear. Headache and vomiting recur, the child becomes restless, anxious, tosses itself about from side to side, crying and moaning; the tongue shows a smeary thick coating, the breath has an ammoniacal odor and in the expired air ammonia can be demonstrated; appetite has disappeared completely. Frequent desire to micturate and fecal evacuations occur, but not a drop of urine can be pressed out. Gradually this condition of excitement is succeeded by somnolence, the child is quite apathetic, with closed eyes; it snores and if awakened, rapidly returns to a state of sopor as if under the influence of alcohol, or it is even entirely unconscious, and now the picture that has become monotonous is varied by the sudden appearance of general spasmodic attacks resembling epilepsy. Sometimes this condition is preceded by partial, even quite monoplegic spasms, for example, in one half of the arm or one half of the face, and then only does the major attack, with general tonic-clonic spasms, pupillary rigidity, foaming at the mouth and complete unconsciousness take place. These attacks may succeed one another until a rapid fatal exhaustion occurs. All the spasms cease after a number of attacks and consciousness returns. Then those about the child are appalled by a new phenomenon, the child has lost its sight. The appreciation of light and darkness may have ceased entirely. This uremic amaurosis is not due to any change of the eye-ground but it is to be looked upon as a toxic disturbance of function in the light centre of the cerebrum. This is favored by the rapid disappearance of this pathologic symptom as well as by the observation that the amaurosis before it disappears entirely, sometimes changes into hemianopsia.

This convulsive catastrophe sometimes has the appearance of an actual

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<sup>1</sup> *Deutsche med. Wochenschrift*, 1902, Nr. 9.

crisis. Soon after the disappearance of the severe spasms the quiescent urinary secretion returns, a highly intense bloody urine is voided in rapidly increasing amounts, the blood contents then disappear simultaneously, the other threatening symptoms abate and the patient recovers. In some few cases there is added to this acute attack a prolonged permanent condition of psychical disturbance, a melancholic depression, with renewed spasmodic attacks. This, however, occurs almost always only in individuals with a nervous taint.

Unfortunately, often enough, even during the attack, death occurs.

The material or the materials which produce this influence of a true intoxication upon the cerebrum, giving rise to this symptom-complex, in spite of numerous investigations, have not yet been determined with certainty. That this intoxication is due to a retention of products which have not been excreted can scarcely be doubted. It may, however, be remarked here, that the severity of the uremia in all cases is not parallel to the degree of anuria. I saw a fatal case of uremia (in severe scarlatinal nephritis), in which the spasms, that in a few hours caused death, occurred upon the afternoon of a day in which the secretion of urine amounted to 600 c.c. Two days previously, however, the uremic headache had already appeared. Just this, in my opinion, is in favor of the fact that scarlatinal uremia is not alone due to action of the retention of a product of excretion known to us, but to the formation of new toxic products, the retention of which in a number of regions of the kidney is sufficient to give rise to severe even fatal damage to the nervous substance.

Strauss,<sup>1</sup> in his examinations of the blood of uremia in chronic renal cases, appears to have come to a similar conclusion when he says that "the increase of the molecular concentration of the blood is an accompanying symptom, *not the cause* of uremia, that the poison which produces uremia is mostly found in those persons in whom also other pathologic substances are found in the blood in abnormal profuse amounts." But, naturally, uremia is usually to be the more feared, the more marked and continued the diminution of the excretion of water, because in this case the substances which are soluble in water remain in the kidneys.

This condition, parallel with the excretion of water, is shown by a second symptom which is a regular accompaniment of severe scarlatinal renal inflammation: *dropsy*.

Although it is not entirely absent in mild cases, this symptom only becomes intense when combined with a low excretion of urine. The face, legs and arms swell, the abdominal covering becomes markedly edematous, as well as the scrotum and penis. At first the swelling is firm, so that the pressure of the finger soon disappears, but after a prolonged existence it becomes flaccid and soft as in other cases of anasarca. To this must be added the transudate into other parts of the body, ascites, hydrothorax, hydropericardium, and

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<sup>1</sup> "Die chronischen Nierenentzündungen in ihrer Einwirkung auf die Blutflüssigkeit." Berlin, Hirschwald, 1902.

in some cases very serious pulmonary edema and edema of the glottis. Regarding the development of dropsy, much debate has arisen. It was believed that simple retention of water was not sufficient to explain anasarca, there must be added especially an alteration of the vascular walls. In fact, I recently noted in the skin of a dropsical child inflammatory changes about the cutaneous vessels similar to those noted in recent cases of scarlatina. But whether this is necessary, is very questionable. The water that the organism does not succeed in excreting must remain somewhere, as the lungs are not able to dispose of it; but, while it cannot be denied that dropsy may occur with a plentiful secretion of urine, being absent upon a diminution lasting for any time, the latter condition refers to a very few days, and in the former circumstance another point must be considered, to which various authors have already called attention (for example, Fürbringer), the importance of which must not be undervalued.

Scarlatinal nephritis also has an effect *upon the heart*. There are not many diseases which, like scarlatinal nephritis, even after a brief existence, are capable of bringing about a decided dilatation (and under some circumstances also hypertrophy of both ventricles). This action upon the heart muscle not only can be proven at the autopsy, but shows itself by a dissemination of cardiac dulness, by the diffusion of the apex beat and the influence upon the pulse (at the onset often abnormal slowing, see above, later the pulse wave becoming small and low upon increased cardiac action), but also by very conspicuous clinical signs denoting a diminished function of the heart. Then there develops, often only after a successful recovery from a uremic catastrophe, a new series of difficulties of another kind. The child again becomes restless, cannot sleep, loses its appetite, begins to complain, this time, however, not regarding headache and nausea but of a tightness and pressure in the chest, of dyspnea and cardiac anxiety. Now the child becomes dyspneic without any cause of this symptom being discernible on the part of the lungs. The condition is one of cardiac dyspnea. About this time, as a rule, dropsy and, later, transudation into the serous cavities begins to conspicuously increase. The secretion of urine, however, does not diminish markedly and the sediment shows a great tendency to the formation of urates. This new increase in dropsy is apparently not only in connection with the disturbance of the renal function but for a large part it is due to cardiac weakness. Thus we meet here for the second time (or including endocarditis, the third) with a danger which threatens the heart, due to scarlatina.

These infrequent cases, in which scarlatinal nephritis leads to a marantic condition, therefore to a general high-graded dropsy, enormous swelling, especially of the scrotum, deathly pallor, complete anorexia, sometimes accompanied by profuse diarrhea, cool and cyanotic extremities, scarcely perceptible pulse, producing a pitiful condition which often drags along for many weeks and then, on account of many varieties of complications, bedsores, phlegmons, gangrenous erysipelas, pulmonary hypostasis, brings about the fatal termination—find their explanation particularly in the severe damage to



the cardiac power. Naturally, the phenomena of chronic uremia may increase the difficulties. But even during the first weeks of a renal inflammation the symptoms of decided cardiac asthenia may be intermingled with those of uremia. Such cases, in my opinion, always show a very serious prognosis. An important sign of this combination is *the increase in size and tension of the abdomen* which may be determined in some anuric patients. This is due to a high-graded enlargement of the liver in consequence of stasis, and this again is a consequence of the failure in the circulation of the blood.

The cause of the fatal termination of anuria is not always the nervous system. A child that was recently observed by me, in which anuria existed nine days, sat in its bed, played with its toys, was restless from time to time, but in most respects did not present a condition that would cause an inexperienced person to suspect the terrible danger which existed for the little patient. Not a spasm occurred before death, and the mind was clear. Finally, in the course of a few hours the symptoms of slight stenotic respiration appeared (consequent upon edema of the glottis) and then the child rapidly succumbed in a severe collapse. In this instance apparently not the usual uremic cerebral intoxication, but a cardiac intoxication was the decisive cause.

Finally, the same condition must be assumed, in which the fatal termination of severe nephritis is brought about by *pulmonary edema*; on the other hand, the addition of a pulmonary affection is to be looked upon as a dangerous secondary mixed infection.

In a case of this kind observed by me, in a boy aged one year and a half, the renal inflammation occurred on the thirteenth day of a medium severe scarlatina; upon the fourth day after this the secretion of urine fell to 50 c.c. per day and at the end of the second it again rose from 50 to 100 c.c. without uremic symptoms appearing. In the beginning of the third week, in the decidedly dropsical child, a high irregular fever and a pulmonary inflammation occurred, during which the edema almost completely disappeared. At the end of the third week of the nephritis death occurred, being ushered in by tracheal râles. The autopsy showed enlarged kidneys, the parenchyma of a yellowish-red consistence; hypertrophy of the left ventricle; lobar pneumonia of the right lower lobe.

If all the dangers are considered which threaten the life of a case of scarlatina complicated by renal disease, we are actually astonished that the prognosis in scarlatinal nephritis in general is not so serious as might be supposed from the first view of the case, that, moreover, upon the average six-sevenths, in many epidemics still more patients, recover from this condition.

It is true, whether acute nephritis is mild or severe in its onset, in the background there still hovers for the apparently convalescent patient a threatening spectre: the transition of the acute into a subacute and chronic affection.

Formerly this eventuality was scarcely considered, denied by some physicians, or at least looked upon as very exceptional. Since convalescents are constantly examined and for weeks kept under observation, and the renal secretion investigated, we have become convinced that this development is by no means so rare, even though the children subjectively and—apart from

the composition of the urine—objectively, scarcely show disturbances in their general health. Often enough, however, in the further course of the affection a decided rekindling of the disease interrupts this condition, which then takes a similar course, giving rise to dangers like those of the first onset of the affection.

Usually the transition into this permanent condition occurs in the manner that after the complete disappearance of the dropsy, and of the possible uremic symptoms, and after the reappearance of a profuse urinary secretion, the chemical examination of the urine shows a permanent excretion, often of scant, frequently also of profuse, amounts of albumin. This excretion of albumin may often show a distinct *orthotic character*, i. e., it disappears when the patient is in the recumbent posture and reappears as soon as he assumes the erect posture. Later it may, however, lose this character. The color, weight and amount of the urine often show no decided deviations, at most that urine following a period of diminished excretion, this being sometimes succeeded by decided polyuria; but the microscopic examination of the sediment teaches that we are still dealing with a mild hemorrhagic form of nephritis. Besides casts of various kinds, granular, hyaline, waxy, also some containing blood corpuscles, almost without exception some few red blood cells are met with; however, as a rule, many more leukocytes are present; finally, almost constantly either few or a large number of fatty granular cells are found, sometimes free, sometimes adhering to casts. Not infrequently *intermittent albuminuria* may be noted.

This continues for weeks. The weeks become months, the months become years; and if albumin were not present in the urine, the children, who usually only have a pale color and complain more or less of headache, would hardly be considered ill. But the question always arises: What becomes of them? This is certain: In favorable cases, even after an existence for years, the albuminuria may disappear completely; this usually occurs in the period of puberty. A case of this kind occurred in the son of a physician and was determined by the father. Secondly, from a nephritis of this sort, within a few years or after a decade, a complete typical contracted kidney may arise. An instance of the first kind I have observed myself, and of the second Dixon Mann gives a very striking example.<sup>1</sup> It can scarcely be doubted that not all the cases which continue unimproved in youth finally take the course observed by Mann. Thus, we meet here with another danger of scarlatinal infection which casts a shadow over the entire future life of the patient who is fortunate enough to recover from scarlatina. For although such patients during the entire life may show a certain degree of health they hardly ever reach old age.

[Such persons show a remarkable tendency to develop albuminuria (sub-acute or acute nephritis) upon exposure to dampness, especially dampness

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<sup>1</sup> Heubner, "Chronische Nephritis und Albuminurie im Kindesalter." Hirschwald, Berlin, 1897, pp. 54 u. 52.

associated with cold, or upon the occurrence of any acute disease. I have for some years been in the habit of interrogating patients suffering from renal disease, in regard to scarlet fever, and in particular post-scarlatinal dropsy in their childhood, and have been astonished at the frequency of the association of these conditions in individuals who have for many intervening years enjoyed excellent health. A much larger proportion of those who develop nephritis in adult life give a history of severe scarlatina with or without dropsy in childhood than a history of the milder forms. But there are mild forms which cause nephritis as a direct sequela, and a chronic form lasting decades, ending sometimes in sudden death preceded by a convulsion.—EDITOR.]

There are still some complications, which, although not belonging immediately to the clinical picture of scarlatina, must be considered as they sometimes influence the entire course of the affection.

Little has been said regarding an *implication of the respiratory organs* in the affection. The diphtheroid mucous membrane affection of these, even in severe cases, does not pass beyond the larynx. The ominous importance of hoarseness and the stenosis which appear have already been noted. But occasionally there is added an intense purulent inflammation of the bronchi and lungs which may reach such a degree of severity that this alone is sufficient to cause the unfavorable termination.

Recently, in the clinic, I saw a boy aged five years who showed high fever, while the eruption was still at its acme. He succumbed upon the sixth day of the disease, showing severe changes in the bronchi and lungs. The palate was hardly red, there was only medullary swelling of the lymphatic tissues, neither the tonsils nor the larynx being implicated; however, the lower lobes of the lungs upon both sides were of tough consistence and of a dirty brownish-red color. Upon section, a bloody edema was noted, but air was also present and from all bronchial openings fluid pus escaped. Upon both pleura fresh fibrinous deposits. The heart was very relaxed.

In a second case, in a boy of ten, who died on the fifth day of the disease, there was found in the left lower lobe, a disseminated purulent capillary bronchitis, lobular infiltration and edema.

I once saw in a nursling of six months, upon the sixth day of scarlatina, the appearance of a severe capillary bronchitis, from which, however, the child recovered.

The bacteriological examination of the bronchial mucus of the cases occurring in the clinic, in which bronchitis was present, showed the presence of streptococci, so that we will not be far wrong if at least in the severe forms of bronchopneumonia we regard these cases as due to the aspiration of these microbes which play such an important rôle in the secondary infections in scarlet fever.

The *organs of digestion* are with comparative rarity affected to any marked extent. Sometimes jaundice is noted in scarlet fever; it may be of a catarrhal nature and does not influence the course of the disease in a prominent



manner. I have seen it occur also in septic cases in which, however, it presents an ominous sign.

Severe diarrhea is sometimes met with in septic pharyngeal affections; it only occurs in the severe and usually fatal cases, taking place in the last few days before death, and is due to an inflammatory swelling of the mucous membrane, especially of the large intestine. Here perhaps streptococci are also the cause.

The *central nervous system* in the case of scarlatina, similar to the other acute exanthemata, occasionally presents severe disturbances, which are so closely connected with the infectious disease that it is difficult to speak of them as mere accidental occurrences. A case recently observed by me is an example of this:

A girl aged eight years was attacked upon November 22, 1900, by diphtheria, which was cured by antitoxin treatment. Upon December 2d a new illness appeared which at first was supposed to be a serum exanthem, but soon showed itself to be scarlatina complicated by severe diphtheroid. Great destruction in the pharynx, numerous lymph gland abscesses and nephritis prolonged the disease for over two months. Up to the middle of February, 1901, fever was present; thence on there was steady improvement, with a good condition and a fair appetite. But still a frequent pulse was present; nothing abnormal could be noted in the heart.

Suddenly upon February 19th, early in the morning, with a clear mind, clonic spasms occurred in the left half of the body, followed by slight left-sided hemiparesis and some mental confusion. Upon the afternoon the spasms recurred but were limited to the left extensor digitorum pedis. Upon April 6th, the child was brought to my office, spasms had not recurred, no paralysis, heart normal, but upon April 7th, again left-sided spasms occurred in the arm and leg, which increased from time to time in severity so that in the second half of May they occurred daily. From then until October the spasms did not cease, occurring almost daily, and only by an energetic bromid treatment were they somewhat suppressed. Other disturbances of the cranial function were absolutely absent. Intelligence was retained. Also the power of movement in the left half of the body was but very slightly influenced. Since that time I have not seen the child.

It can scarcely be doubted that here a superficial meningo-encephalitic focus had developed over the right central convolution during the attack of scarlatina. The spasms had the entire character of those occurring in Jacksonian cortical epilepsy.

**Combinations of scarlatina with other specific infectious diseases** occur with great variations.

Especially important is that occurring with *bacillary genuine diphtheria*. Quite often not only in the same locality do scarlatina and diphtheria occur endemically—which has led to the erroneous assumption of a close relationship of both diseases and especially of scarlatinal diphtheroid with true diphtheria—but even in one and the same family scarlatina and true diphtheria may occur side by side. I have noted instances of this in which children suffered from unquestioned scarlatina without diphtheria, whereas

the father simultaneously suffered from bacillary diphtheria which was followed by severe, prolonged paralyses.

If both affections combine in the same organism, it is much less serious if diphtheria is added to scarlatina than inversely. Each year, in the clinic, one or the other case is observed in which genuine Löffler bacilli can be detected in the pharyngeal parts, these areas then for the most part being covered by dense membranes. If the specific treatment is begun at once, the second infection does not materially add to the difficulties of the first, even if the scarlatina should be of a serious character. This I saw in a severe scarlatinal diphtheroid, in which later on, not only new deposits in the pharynx but also the walls of a deep and distributed ulcer cavity in some lymph glands in the neck covered with membranous deposits occurred. Everywhere the typical Löffler bacillus could be demonstrated. After specific treatment, which was employed at once, the diphtheria rapidly disappeared and finally the very ill child recovered completely. Naturally, when the true character of the pharyngeal affection is not recognized, that laryngeal stenosis, descending croup or true diphtheritic paralyses may follow a "scarlatinal diphtheria" is easy to understand, but does not alter the fact that the usual diphtheroid pharyngeal infection has nothing in common with true diphtheria.

The condition is much more serious if a scarlatinal infection is secondarily added to diphtheria. Then it avails but little that the primary affection has been successfully and specifically treated. In the winter of 1894-95, while the curative serum treatment was in full swing in the diphtheria division of my clinic, a scarlet fever epidemic of 12 cases occurred, almost all arising in November and December. The original diphtheria patients for the most part were medium severe cases and were at once treated with curative serum; in spite of this, 3 of the 12 cases died, therefore 25 per cent. The originally diseased diphtheritic parts, after the appearance of scarlet fever, were attacked by a hemorrhagic necrotic inflammation. The transition of the membranous diphtheria to a necrotic and ulcer-forming diphtheroid inflammation could be well followed. The cause of death is always due to sepsis, in one case with the addition of jaundice, in another of the hemorrhagic diathesis.

The combination of *varicella* and scarlatina, especially if the former is added to the latter, runs its course without disturbance. Not rarely is scarlatina added to varicella in the manner that the erythema, as from a wound, takes its point of origin from a scratched varicella pustule. Then the scarlatina does not always run a favorable course but develops a stubborn nephritis or other complications.

Once, in a girl aged nine years, I saw scarlatina develop in connection with varicella. To the scarlatina there was added a severe erythema exsudativum multiforme, to this simultaneously there was also added hemorrhagic nephritis and a severe hemorrhagic diathesis resembling scurvy, with marked hemorrhages from the gums and, finally, measles appeared. But from all these diseases, after an illness of three months, the child finally recovered completely.

Occasionally in convalescence from scarlatina, the appearance of purpura is noted. Two cases which occurred a year or two ago in my clinic, after a few weeks, terminated in complete recovery.

Regarding the complication of measles and scarlatina this has been described under measles (which see under Measles).

**Puerperal scarlatina**, according to the investigations of Sørensen, as well as of other authors, is nothing else but the usual scarlatinal infection which finds entrance by way of the damaged parts. The previously mentioned author found the diphtheroid infection in the perineum and in other wounds of the genitalia instead of in the pharyngeal organs.

In a pregnant woman aged thirty-five (toward the end of pregnancy), upon the third day of the disease, I saw the birth of a dead child; there were no signs of the eruption present. A woman aged thirty-four years who was nursing her child during a medium severe attack of scarlatina kept on nursing without the child being attacked.

## DIAGNOSIS

In well developed cases the scarlatinal rash has a characteristic appearance. The fine, delicate, closely situated elevations seen at the onset of the disease, which, after the general cutaneous exanthem has appeared, remain during the entire course, are not met with in the same manner in other eruptions of a similar nature. At most, the "triangular form" of the prodromal variolous eruption might be assumed which sometimes exactly resembles the scarlatinal exanthem, even including the deep fine elevations, but this is so limited to a triangular space upon the thigh or the shoulder that the differentiation may be very readily made.

But there are acute diseases in the early period of which a *scarlatinoid exanthem*, an eruption, appears simulating a form which is not rare in scarlatina and which may then give rise to grave errors. This is noted in enteric fever and in acute lobar pneumonia in children as well as in adults. General cutaneous erythema even with succeeding mild desquamation particularly occurs in the first days before the previously mentioned characteristic symptoms of the disease have developed. I know of a case in a lady whose severe enteric fever which terminated fatally was incorrectly looked upon as scarlatina by a prominent diagnostician. In children I have much more frequently known the mistaken diagnosis of pneumonia to be made. It will always be well to remember the small prominent points in the exanthem before a diagnosis of scarlatina is made.

Besides there are also toxic exanthems which may give cause for confusion to less experienced physicians; thus, we occasionally see drug eruptions of a scarlet red color produced by quinin and antipyrin, but the injection of the deeper layers which is so characteristic of scarlatina (upon pressure with the finger), the *raie blanche*, and the unevenness of the skin are absent. For



a long time the similarity of the rash of atropin to that of scarlatina has been known, which even once led to regarding belladonna as a preventive against scarlatina. Further, the toxic products contained in the serum of animals often lead to a scarlatinoid eruption. These forms of serum exanthem, especially in the diphtheria departments of hospitals, often are the cause of great anxiety. They may appear with severe fever, with headache and vomiting, and not infrequently are the palatine parts inflamed, even new deposits occurring. Briefly, the entire picture of beginning scarlatina is present. There are cases in which the question whether we are dealing with scarlatina or not, must remain undecided—even the combined opinion of such experienced eyes as those of a Widerhofer and of a Kaposi have been impossible to decide this question at a first glance.

In these cases another property of the scarlatinal rash which makes the diagnosis difficult must be considered: that it by no means always reaches the full development which was previously described as so characteristic. On the contrary, in unquestioned cases it quite frequently remains in a stage of pale, indistinct, rose-colored maculæ, in which confluence does not occur, without attaining a scarlet color during its entire course. Sometimes even distinct spots do not appear but we only find a slightly developed erythema upon the trunk and upon individual parts of the extremities. Exanthems of this kind naturally occur in quite a number of other affections. Apart from the eruption due to serum, we find this in a well developed manner in the scarlatinal form of rötheln. But it will be opposed that this disease runs its course without fever! That would be a decided point of difference if there were not cases of scarlatina without fever, or at least with only a slight initial rise, which may have disappeared at the time of observation. A short time ago, when in consultation with one of the first physicians of Berlin, I had to give an opinion in a case in which he was in favor of scarlatina, while I was in favor of rötheln, without either of us being able to give decisive proofs for his opinion. The further course of the disease favored my opinion in so far as no nephritis or other serious difficulty arose. In these difficulties it becomes quite clear how much the clinician owes to the bacteriologist, in all those cases in which an unquestioned etiological diagnosis is possible. At the bedside, in doubtful cases, it would be well to examine the entire surface of the body; frequently, at least here and there, an area is noted in which the fine papular character of the rash shows the nature of the disease. Also the conditions of the oral and pharyngeal cavities which will be described later may aid in this.

An influenza, also with cutaneous erythema, occurs, which may resemble a feebly developed scarlatinal eruption. A few years ago, in the family of a colleague, I was required to give an opinion in which, simultaneously, fever, and an angina with a beginning exanthem were present; the rash having the character of a fleeting form of scarlatinal erythema. Only the circumstance that almost within five days all the cases occurred suddenly, all the patients being adults, and in a short time recovered without compli-

cations favored influenza against scarlatina. As no expectoration was present no decision could be given by a bacteriologic investigation.

Finally, cases occur in which the exanthem of scarlatina does not present its usual appearance, especially upon some areas of the body, as, for example, the arms and legs—with the trunk remaining almost entirely free—where it shows a coarse macular and partly papular condition, which is common to measles. In these cases the correct course will be indicated by the character of the mucous membrane changes.

The diagnosis may become very difficult if the exanthem shows itself in a rudimentary character or is absent altogether. In the former instance the correct trail can often still be found if it is only not forgotten that the entire surface of the body from head to foot must be minutely examined, especially also the posterior surface of the body, the elbow, the popliteal space, etc.

Where no eruption is present at all, auxiliary circumstances must decide: The circumstances in the surrounding of the patient, the onset of the disease with vomiting, a disproportionately high pulse, etc., and often the later appearance of marked desquamation may show the true significance of the case.

In such cases in which a suspicion is entertained, the condition of the oral and pharyngeal cavities must be observed. The strawberry tongue, the sharply defined, deep red appearance of the velum of the palate, of the uvula and of the palatine arches, the marked deposits on the tonsils, the flow from the nose, the intense swelling and painfulness of the lymphatic glands justify the assumption of a latent scarlatina. The deposits upon the tonsils and the other palatine parts in this form of scarlatina often show an exquisitely membranous character, especially in the first days, so that unless a bacteriological investigation is made, primary diphtheria is much sooner supposed to be present than a diphtheroid. The diphtheria divisions in hospitals are frequently endangered by such cases, namely, by the introduction of scarlatina.

This shows the great practical importance of a correct diagnosis in all questionable cases that have been mentioned, in that the feebly developed, quite rudimentary eruptions, and even those of infections that run a course without rash, are capable of conveying the disease to predisposed organisms, which in the latter may show a severe course and give rise to a fatal issue, and this may occur quite as readily as by the fully developed cases of scarlatina. For this reason the greatest caution is necessary even in every suspicious case.<sup>1</sup>

## PROGNOSIS

The general prognosis of scarlatina depends upon the character of the epidemic, which, as has been expressly mentioned, varies greatly in different

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<sup>1</sup> A generalized erythema may be caused by intestinal autoinfection. Improper food, fat feeding, the "top milk" prejudice, in some even unmixed milk feeding, will cause it. Then indican, skatol, indol are found in the urine, often with more or less symptoms of kidney irritation, microscopic blood, hyaline casts, also fine granular casts. Often increase of temperature.—EDITOR.

periods; also in different countries, and in different races, the prognosis is not the same. In my private practice in Leipzig, lasting for fifteen years, in which I had to do with the district poor, which, therefore, represents the least resistant part of the population, in 358 cases I had a mortality of 13.4 per cent. We must, however, admit in the case of scarlatina that the influence of better conditions of life plays a much less important part regarding the chances of recovery from the disease than, for example, in the case of measles. Jürgensen, in Tübingen, among 547 cases, had only a mortality of 8.23 per cent.; in Stockholm, according to this author, the mortality in different years varies between 2.8 per cent. and 28.8 per cent., upon the average 16.3 per cent.; in England, the mortality varies from 13 per cent. to 40 per cent. In Norway, in ten years, Johannessen found an average mortality of 16.6 per cent. (in children). From these figures so much can be concluded that an individual epidemic, especially in England, may become a calamity almost resembling the most terrible well-known pestilences.

In the individual cases there are very few acute diseases in which it is impossible for so long a time to give a certain prognosis as in scarlatina. This is due to the fact that even in very mild cases the affection at the onset, even at the end of the third week or later, may show a change due to nephritis, which may threaten life or at least produce chronic invalidism. Nevertheless, a regular uniform course—oftener with a well developed exanthem than with a feeble or rudimentary one—during the first week of the disease may be looked upon as a favorable prognostic sign.

Any deviation, no matter how mild, a slight rise in the fever, renewed swelling of the glands, etc., never remains isolated but opens the road to all possible serious consequences. Even in private practice rendering a prognosis is greatly facilitated by the keeping of a regular temperature chart: the fever in this instance is a very accurate prognostic indicator. Especial importance should be given to the second half of the first week, in which the first signs of diphtheroid or of otitis media become noticeable, and to the turning point between the second and third weeks, in which nephritis is most liable to occur.

For the prognosis of a severe case with cranial manifestations, three symptoms in particular appear to me to be especially unfavorable: jactitation, dyspnea, with loud respiration, and abnormal frequency and smallness of the pulse (as well as the other signs of cardiac asthenia). Severe stupor, or marked delirium are in themselves by far less dangerous phenomena.

In diphtheroid, the unusually marked enlargement of the glands and the coarse periglandular infiltration which occurs early are very unfavorable prognostic symptoms, as are also a very markedly rising fever, or an otitis media; the prognosis goes hand in hand with the severity of the fever in general.

Finally, in nephritis—at least in most cases—the most certain indicator of immediately threatening danger is the decreased quantity of urine excreted. By the mere estimation of the albumin and also by microscopic investigation of the sediment by no means so accurate an opinion can be gained in regard



to the condition as by measuring the daily quantity of the urine. For this reason, even in private practice, we should in no case of nephritis neglect to measure the quantity of urine for each twenty-four hours.

Finally, the age of the patient is not without importance in the individual prognosis. In the interval from the first to the fifth, and especially from the second to the fourth year, life is mostly threatened by the manifold dangers of scarlatina.

## PROPHYLAXIS AND TREATMENT

The prophylaxis of the disease consists chiefly in the prevention of contact between the sick and the well in so far as is possible, and only the diseased human being presents the danger of further distribution. The attainment of this end should be worked for, as the scarlatinal infection need not attack every person who is exposed to it, for the susceptibility to the poison decreases with advancing years and, on the other hand, every infection may lead to a malady dangerous to life. This course is more easily prescribed than carried out, as, especially in families with many children closely crowded together, the separation of the sick from the well is often quite illusory, but even among the better situated classes of the population there is great difficulty with this prophylaxis, as the disease, especially in adults, appears in a form which is not readily recognized, showing but the usual angina, but which fact does not materially interfere with the transmission of the disease. Just this circumstance, however, makes it our imperative duty to prevent contact of those around the scarlet fever patient with others free from the disease, thus, they should not be allowed to go to school, to infantile institutions, kindergartens, hospitals, etc. Difficult as it may be for the affected family, the brothers and sisters of the scarlet fever patient must remain isolated during the entire time of the disease. In many countries this is required by law.

The question is often asked of the physician whether the healthy nurses or relatives of the child may visit other families, may receive company, travel, etc. Particularly in regard to the possibility of the transmission by the healthy, or at least apparently healthy, I would advise prohibiting this. Transmission by the physician from one sick-bed to another or to his own family is not likely as the visit of the physician to the bedside is usually a very brief one and a sufficient quantity of poison does not adhere to him. If only he does not have a latent, unsuspected scarlatina himself! Yet the greatest cleanliness and care is absolutely necessary even on the part of the physician. Wherever possible, his visits to patients of this kind should come at the end of his visiting list, and the custom which is coming into vogue of covering the clothes with a linen gown in visiting the sick should perhaps be generally introduced.

Particularly difficult and important is the question of disinfection of the sick-room after termination of the disease. This involves great inconvenience and often enough decided injury to the infected individual, and also in not

a few cases its value has been shown as illusionary, and we must actually admit that it has not been strictly proven that the poison of the disease may adhere to the sick-room in a condition capable of further dissemination. In most cases of this kind, when the disease has terminated (for example, by death) and the poison has still shown itself to be active, there remains the possibility that it has been present in the oral cavity of a person who has been active about the patient. Nevertheless, in view of our present knowledge of the nature of the scarlatinal poison, it is perhaps impossible to avoid unnecessary measures.

All these difficulties could to a great extent be avoided if in every case of scarlatina (as also in other infections), instead of sending away the well children as is usual, the entire population would agree to send away every sick child after the disease has been determined with certainty. In order to carry out this plan it would be necessary to have more well-equipped hospitals for treatment of contagious diseases than exist at present, as do some other countries (America, England, Denmark, Sweden). Where these opportunities exist they are at present largely shared by the rich and the poor, this being shown by the experiences in Stockholm, with the new infectious hospital erected there, and I have also observed this since the erection of the new Children's Hospital in Leipzig. With increasing frequency, the first families of the city come with their children sick of scarlet fever or diphtheria to the hospitals to have them treated there. Especially in regard to the better situated classes, it is necessary, in the erection of such hospitals, to make it possible that the mother may accompany her child. Yes, even more. The families should not be required in such cases to give up their physician who has perhaps been attending them for years for one who is unknown to them, nor should the dignity and position of a thorough family physician be sacrificed by allowing his patients to pass into the hands of any other physician. I see the solution of the dilemma only in this, that, according to the example of the many sanatoria which exist in large cities, in which infectious patients are taken, every practising physician should be allowed to continue treating his patients with or without the cases being accompanied by relatives. Nothing is changed but the sick-room. Transportation among the first hours or the first few days of the disease is always possible. In the course of time a system of this sort is bound to develop.<sup>1</sup>

That, naturally, there should be a disinfection of all utensils and objects which have come into contact with the patient, bed, toys, etc., which may be contaminated with the poison, and are, therefore, dangerous, is clear. They should be disinfected and if possible destroyed. It is impossible to enter here more minutely into these hygienic questions.

Simple scarlatina has a typical course which, so long as we are not in possession of a specific remedy, had better not be disturbed by interfering

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<sup>1</sup> Without any criticism of our defective American conditions, I merely wish to direct the attention of the reader to these lines.—EDITOR.

measures. The object of the physician consists in the widest sense in ordering the diet and in superintending the nursing. The patient in most cases goes to bed of his own accord and even in the milder cases should be kept there for at least three weeks and not allowed to get up, until by a careful examination of the urine which has been saved for twenty-four hours it has been shown that the kidneys have been spared. This indication should be carried out under all circumstances.

Wherever possible—even in small dwellings—the patient and the nurse should each have a room. The rest of the family during such an unfortunate time must make up their minds to suffer some inconvenience. In better situated circles wherever possible two neighboring rooms should be used. As soon as the disease has been recognized the physician must prevent the other children from going to school. What a calamity this is, in the narrow rooms which often represent living room and kitchen in workmen's dwellings, may be imagined. I have had some experiences which would scarcely be believed. Therefore, in such instances it is our duty to use all our influence toward sending the sick child to a hospital. Naturally, often enough the money question is in the way, provided the case occurs in a family that has recently come to the city (in Germany). The patient must not be allowed to have company.

The sick-room is to be kept rather cool than warm, from 64° F. to 68° F. is sufficient. The covering for the fever patient is to be light. The child is to be supplied with fresh body linen and bedclothes, which during the disease must be carefully (previously warmed) renewed. The nutrition in the first three weeks should consist of a pure milk diet which may be varied in many ways (adding some coffee, bonnyclabber, buttermilk, rice and milk, milk and grits). The assumption that by these means the development of nephritis may be prevented is erroneous, but it is possible that by a too early administration of food containing extra amounts of albumin the kidneys may be irritated and nephritis caused in this manner.

The skin as well as the mucous membranes must be kept scrupulously clean; wherever possible, and especially if the children are accustomed to it, the patients are to have a quick luke-warm bath daily. In other cases the face, hands and feet, and any soiled parts of the body are to be washed with luke-warm water and soap. Especial attention is to be given to the daily cleansing of the nose, by cotton tampons which have been moistened, and the careful washing of the mouth and pharyngeal cavity, and by frequent active or passive cleansing, such as gargling with a luke-warm, dilute salt solution. This is to be repeated five to six times daily. It is advisable to use cheap substances such as cotton or the like, which immediately after use may be collected and burned.

For the thirst, dilute acid drinks, lemonade or the like or boiled water with a little citric acid and sugar, are given. Internal medication is quite unnecessary. I am decidedly opposed to the early use of antipyretics, those from the group of benzol derivatives. Only apparent success is attained and



the regular course of the disease is disturbed. Where there is a necessity for influencing the fever, a plentiful amount of cool drink (but without the addition of alcohol) should be administered, and there may be cool applications of towels doubly folded and moistened and applied upon the chest, abdomen, and thighs, these being covered by woolen cloths. These applications may be readily fastened around the abdomen by means of bandages. In marked coma and severe delirium one or two, perhaps more, luke-warm baths are given at a temperature of 91.5° F., with cold affusions. The technique has been described in the treatment of measles. During the period of desquamation, this is favored by luke-warm cleansing baths. In cases in which there is marked itching or burning, inunctions with washed lard which were formerly so much used may be employed or a 1 per cent. thymol-lanolin salve may be of value.

Thus, convalescence is awaited and only the course of the temperature is carefully observed so as to be prepared for threatening danger.

If such dangers develop, prompt interference is necessary even in desperate cases, if only to give the parents the satisfaction of feeling that no measure that is known to professional skill has been neglected in the attempt to save life.

Before entering upon the discussion of the treatment of the individual varieties of anomalous scarlatina, a few remarks regarding *general treatment* which influences the scarlatinal process may be in place.

The most important is the attempt to introduce a serum therapy. This depends upon investigation showing that the recovery from scarlatina confers immunity toward the infection for the one that has recovered; this immunity is referred to the presence of antitoxins in the blood of the convalescent. As well as in the case of the animal that has been inoculated with diphtheria, in which the antitoxin-containing blood serum shows a curative action, so also the serum of a scarlatina convalescent is said to remove the dangers of blood intoxication due to the scarlet fever attack. It is clear that this serum therapy is erected by hypotheses and should not be mentioned in the same breath with the accurately determined diphtheria serum therapy which has been proven by experiment. We are not familiar with the scarlatinal poison, we do not know whether it forms toxins, nor whether the scarlet fever immunity is due to the permanent presence of antitoxins in the blood. And even if this were the case it would be very questionable whether small amounts of the blood of convalescents which contain so much antitoxin would combine the hypothetical poison in a second organism. It must be remembered that in the case of diphtheria, the animals which furnish the antitoxin have been exposed to an enormously increased severe infection before their blood shows curative properties. Thus, theoretically, the thought of serum in the therapy of scarlet fever in its present condition does not promise very much. I should believe that trials would only be justifiable in those desperate cases in which all other previous measures have proven ineffective. In these cases every rational attempt is permissible.

Another general treatment has recently been advised by Seibert<sup>1</sup> in New York: Repeated inunctions of the entire body several times daily with a 5 per cent. to 10 per cent. ichthyol-lanolin salve. Every six hours the entire surface of the trunk and the extremities is, with slight pressure, to be anointed; in each inunction, according to the size of the patient, 30 to 90 grams are to be used. I have employed this method quite frequently, most children bear the inunctions very well and sometimes it appears to influence the temperature and general condition of the patient. The method is quite difficult, requires much body linen and is very expensive. Of actually severe cases, none have been saved by it, for this reason I have abandoned the treatment. Kraus reports from Ganghofner's clinic, rather an exacerbation of the cutaneous affection by the ichthyol inunction. I have not observed this.

Nor have I seen success from inunctions with Credé's argent. colloidal nor from the subcutaneous use of Marmorek's streptococcus serum. Wherever the treatment has been carried out, it was unsuccessful in a number of cases before being abandoned.<sup>2</sup>

Thus, until now the hope of a successful treatment of the *indicatio morbi* has not been attained.

We shall now turn to those measures which have shown themselves useful in the individual cases in which complications have arisen.

The treatment of scarlatina gravissima, according to my experience, is entirely hopeless. I have never seen a case recover in which the diagnosis was at all certain; the strongest analeptica (up to 1.5 grams camphor subcutaneously in twenty-four hours), antipyretics, hydrotherapeutic measures, are of no avail here; nevertheless, in every instance the attempt must be made to combat the disease with all the energy at our command, and, besides, some cases which are characterized by especially intense fever and severe nervous symptoms resemble the severest form, although they are favorably influenced by the remedial measures now to be named.

Above all, the cooling and stimulating effect of the water treatment must be considered. The very rapid rise of the body temperature to a great height, which in itself is dangerous, requires a purely symptomatic withdrawal of heat. I cannot advise the employment of cold baths for this purpose. I used them formerly but I find that the infantile organism upon the whole does not bear them well, and that in the case in question, in which there is almost constantly a cardiac weakness present this is a contra-indication. I prefer for this purpose repeated packs, the influence of which may be extended to an hour, without causing a decided internal congestion of blood as is the case with a cold bath. For this purpose two beds, each supplied with a sheet and woolen blanket, are necessary. The cover and the sheet, which have been wrung out in cold water (at 59° to 60° F.), are spread out, the

<sup>1</sup> *Jahrbuch für Kinderheilkunde*, Bd. li, p. 308, 1900.

<sup>2</sup> I have a strong impression that a few of my very bad septic cases recovered under the use of the serum, "*Remedium anceps melius nullo.*" A doubtful remedy is better than none at all.—EDITOR.

naked child is enveloped up to the neck, first in the sheet then in the woolen blanket, and allowed to remain so for ten minutes (or if the hyperpyrexia is not so marked, for fifteen minutes). In the meantime the second bed has been prepared in a similar manner and the child after it has been taken from the first pack is immediately enveloped in the second for ten (up to fifteen) minutes, and so forth for an hour, so that the decided cooling is repeated from four to six times. The difference between this method of cooling and a cool bath are obvious enough; between the renewed coolings the sheet warms itself inside of the woolen covering and the blood remains upon the surface of the skin. The result of the cooling is very energetic. This procedure must not be employed more frequently than two, or at most three times a day. In the intervening periods the child is to be allowed to rest.

Secondly, in those cases in which there is marked nervous implication, *cold affusions in a warm bath* are of use. The duration of this (temperature 90° to 95° F., according to the condition of the pulse) may be five, ten, or even fifteen minutes (in older children), but may also be very much shortened. The principal indication here is the cold affusion. The children are comfortable in the warm fluid, which, compared with the temperature of the body, is always 12 to 15 degrees lower, and now the beneficial contrast for the nervous system, the cold, "the cold shock" occurs. From a slight height water cooled by ice is poured over the head, neck, back, and chest, in short intervals according to the length of the bath; at least 5 to 6 litres are to be used. The external auditory meatus is to be closed by plugs of cotton. The parts of the skin upon which the cold water is poured are to be gently rubbed during this procedure as well as the trunk and extremities which remain in the bath. Each affusion causes deep respiration that cannot be attained by any other method, therefore, a very decided area of the lungs is influenced. Quite a number of other reflexes also occur. The effect upon the centres in the medulla cannot be mistaken. The baths are repeated four, even six times daily. Usually after this procedure children will take a larger quantity of nourishment, which is followed by quiet sleep.

A third process has for its object combating the cardiac weakness. There, in my opinion, alcohol is absolutely necessary. Strong, old red wine, and especially champagne, stimulate the cardiac and vasomotor activity so long as the greatest danger appears to be present. After this has passed the alcohol may be discontinued.<sup>1</sup> But during the storm of the life-threatening symptoms, it is well not to be too sparing with it. Other analeptica are to be utilized at the same time, especially camphor, which in an oily solution may be given hypodermically every two hours or even oftener. Of a 20 per cent.

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<sup>1</sup> It should not be discontinued too early. It is the best remedy in sepsis. While this lasts no dose is too large. No intoxication by alcohol takes place during bad sepsis. The best oil to dissolve camphor in is that of sweet almonds. The useful, very useful, preparations of caffein are the double salts: Sodio-caffeïn salicylate or benzoate. They are easily dissolved in 2 parts of distilled water. Fifteen minims contain 8 grains of the salt, equal to about 4 grains of caffeïn.—EDITOR.



solution, according to the age, I give from one-half to an entire Pravaz syringe-ful per dose.

By French authors (Moizard) the subcutaneous injection of caffein (10 per cent. solution of sodium salicylate of caffein, one-third to a syringe-ful to a dose) and where the nervous symptoms, besides asthenia, are especially developed, sulphate of spartein (4 per cent. solution, one-third to one-half syringe-ful to a dose) is advised. I have had no personal experience regarding these procedures.

The treatment of *diphtheroid*, above all things, requires a frequently repeated cleansing of the oral, nasal and pharyngeal cavities. Even the simple, frequently repeated drinking of pure water, or water containing some acid fruit syrup, is of value; but drug treatment must be added. There are a large number of disinfectants which may be used in solution, as a gargle or mouth wash, or may be insufflated into the mouth and nose. Boric acid (5 per cent.) is a favorite, but is not especially active, also salicylic acid (1 to 100), or hydrogen peroxide (3 per cent.) may be employed. In the last few years ichthyol has appeared to me to be of especial value. I use it almost exclusively in the form of a 5 per cent. solution of ammonium or sodium sulphoichthyolicum. I have used it much in private practice and have heard many physicians praise it. The disagreeable taste of the solution is unpleasant, so that some very sensitive children object to it; usually, however, this repugnance can easily be overcome upon the second day. The application occurs in older children in the manner that after washing or gargling with pure luke-warm water (to remove the mucus masses or remains of food) the same process is repeated with the ichthyol solution. In younger children the same object is accomplished by means of a tube, syringe or irrigator. This process has the advantage that under all circumstances the disinfecting fluid also reaches the posterior and lateral pharyngeal wall, which is not the case with simple gargling.

The syringe must not be used through the nose, but a small quantity of the disinfecting solution may be poured into each nasal opening by means of a spoon or the like, it is then allowed to flow out by the mouth; small quantities are naturally swallowed. Or a tampon of cotton, having been dipped in the fluid, is pressed out in a similar manner as was described in the chapter on the treatment of measles. Previous to this, each half of the nose is to be cleansed, as carefully as possible, by means of small pellets of cotton. For nasal disinfection, insufflations are also valuable, for example, of the sodium sozodol powder mentioned under measles. By these remedies the surface of the diseased mucous membrane may always be denuded and thus prevent the increase of the dangerous streptococci.

If, however, necrosis of the mucous membrane has occurred in extended areas in the naso-pharyngeal space, these septic microbes are no longer in reach of superficial washings for they are already proliferating in the deeper tissues.

We even now possess a method of reaching them or at least of causing

some attenuation, even if not destroying them, in the subcutaneous injection of antiseptic remedies into the palatine areas, a 3 per cent. carbolic acid solution being best for this purpose. The object of this treatment is not to influence the diphtheroid locally but to introduce these solutions into the roots of the lymph vessels which lead to the lymphatics of the lower jaw. The antiseptic is to take the same road which the mass of septic streptococci takes as well from the nasal as from the oral cavities, and to meet them in the lymph vessels and lymph glands and render them harmless. The proposal for this method of treatment, which I mentioned in 1886, has been accepted by a number of pediatricists, who like myself were satisfied by the results attained. Its general introduction into the treatment of scarlet fever has not yet taken place, but I have used this method for over twenty years and have not given it up, for it appears to me that it is more valuable than anything else that has been tried. For the injection, a Taube cannula is employed; this is attached to a Pravaz syringe, injections being made twice daily into each half of the palate (tonsil and anterior velum of the palate, arch of the palate), half a syringeful being employed, therefore, in all 0.6 phenol daily. The manipulation in this process is easier and simpler than the previously used method of making applications to these parts. If a brown color of the urine occurs this treatment is stopped. The method is begun as soon as the rise in the temperature, which was mentioned in the clinical description, takes place, upon the fourth or fifth day, or an increase in lymphatic enlargement, etc., announces the danger of diphtheroid; under some circumstances, in severe cases this treatment may be begun upon the first or second day. It is continued until the glands become smaller, the fever declines or the condition of the oral and nasal cavities shows a tendency to limitation of the inflammatory necrosis. Not rarely more or less extensive necroses are found in the surroundings of the openings made by the needle. These are not caused by the carbolic acid itself, but are due to the severity of the local affection and may appear in the same intensity in areas in which no injection has been made. If necroses occur in wide distribution around the point of insertion I stop the injection in the infected half of the palate. In themselves these losses of substance, as has already been explained, are rarely of danger.<sup>1</sup>

The inflammation of the lymph glands, which very often takes a substantive character, may be controlled at the onset by the application of ice. If, however, marked enlargement, hardness, or periglandular infiltration have taken place, then a warm poultice is more valuable to produce suppuration as early as possible so that an incision may be made. The incision of non-suppurating necrotic glands as well as the excision of numbers of such infectious

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<sup>1</sup> I strongly emphasize the warning against the use of syringes. Irrigations of the nose from a cup which reach the fauces, of saline solution, of boric acid solutions, of corrosive sublimate (1 in 5,000 or 10,000, mainly the first) are curative and preventive. They may be made often, every one to three hours. No use in destroying the child's muscular and cardiac powers by the struggles caused by forcing the mouth open. The mouth is accessible by the nose.—EDITOR.

glands has as yet not been followed by favorable results. Regarding inunctions of mercury salve, of argent. colloidal, I have seen no other results than that they make the skin dirty and not infrequently produce excoriation. I leave the cutaneous coverings of these septicly affected parts alone. Analogous to this a purulent parotitis should be treated. I have never seen this complication in scarlatina.

Of greatest importance, besides the local treatment of the scarlatinal diphtheroid in the mouth and nose, is the attention which must be paid to the condition of the middle ear. The same is true here as was mentioned in the discussion of measles, which was accurately described and for this reason need not be repeated.

*Scarlatinal rheumatism* is treated with the same symptomatic results as is polyarthritis, by antirheumatic remedies. I have not seen a justification of the fear that by their internal use the kidneys would be injured. Among 29 cases of scarlatinal rheumatism in my practice, 3 later suffered from nephritis. This is exactly the same percentage which was observed in the total number of cases. At that time the rheumatism was almost exclusively treated by salicylic acid. Now it is better to use the milder and more pleasant *aspirin*, but not in too small doses (afternoons at two, four and six o'clock doses each of 0.5 [ $7\frac{1}{2}$  grains]). As a rule two days of treatment is sufficient. The less severe cases may be treated by keeping the affected joints warm, the symptoms disappearing from them in from four to six days.

*In postscarlatinal fever*, the ear and the posterior pharyngeal wall are to be carefully examined, regarding a possibility of absorption of septic material, the treatment being accordingly. Ichthyol washings of the posterior pharyngeal wall from the mouth render good service.

*Scarlatinal typhoid* is to be treated in the same manner as a mild attack of infantile enteric fever. There should be employed cool packs to the chest and abdomen, cool drinks, under some circumstances baths and drug antipyretics, but here, above all, quinin may be utilized.

*Scarlatinal nephritis* in a great number of cases may take the simple course described and in the main requires but simple dietetic treatment. It is good practice so long as the amount of urine does not fall below 500 to 600 c.c. to desist from active treatment. Milk diet is continued; and by all possible auxiliary measures (addition of some coffee or tea, with variation in the form of buttermilk, etc.) an attempt must be made to give as much of this food as possible (2 to 3 litres), so that the requirements of the body are completely covered. Besides milk, luke-warm drinks may be given (lemonade, tea) or some alkaline mineral water, but if at all possible there should be no other food substance so long as a sufficient quantity of milk can be taken. In those cases in which milk cannot be administered, which are not frequent, the food should consist particularly of vegetables; wheat bread with butter, dried or fresh vegetables, fruit soups, cereals, fruits. Many of these substances may be prepared with milk and will be taken by those that reject pure milk. But the action of a mild diuretic, which milk if it be taken alone shows,



cannot be attained in this manner. Occasionally small quantities of milk will be taken frequently instead of large quantities at one time.

The fever which occasionally accompanies nephritis need not be especially considered in the treatment, particularly as it is of brief duration.

As soon as the urine secretion falls below 400 c.c., showing a more decided injury to the kidney, active measures are necessary. With regard to the marked implication of the vascular system in the damage to the scarlatinal kidney, I hold that every measure which may produce an irritation is a two-edged sword, and prefer for this reason to spare the kidney in the acute stage. Even mild drug diuretics, as for instance potassium acetate, I do not employ, for I allow this function to be carried out by food and by drink. I prefer to make the skin active and to withdraw water by means of sweating. The best method consists in placing the patient in a warm to a hot bath (95° F. gradually rising to 100.5° F.), this lasting from ten to fifteen minutes, and being followed by dry or moist packs. In cases in which fever is present the sheets may be dipped in cool water so that the heat may be withdrawn at the same time and even the bath may be cooler. The patient is allowed to remain one-half hour in the pack after profuse perspiration has appeared in the face. Then the covers are gradually removed, the skin is dried with warm cloths, and the patient is placed in the bed which has been previously warmed. This process is only to be carried out once daily. There are, however, children who in this manner at least cannot be caused to sweat, especially in the beginning, then warm tea may be taken during the pack; and in cases in which this does not act I have very often with advantage given a little pilocarpin. The subcutaneous injections of this remedy have been quite properly abandoned, but small doses internally appear to me to be but slightly if at all dangerous. At the onset of the pack the child is given 10 grams of a solution of 5 centigrams of pilocarpin to 100 grams of water. Almost always, shortly afterward, slight vomiting and salivation occur and the enveloping material should be protected by a cloth which is placed before the mouth; soon afterward, however, sweating takes place. Frequently this method need be used but once, sweat following later without the use of the drug.

In a favorable action 300 grams of fluid may be withdrawn from children, frequently, however, not more than 60 to 100 grams. As, however, according to Strauss,<sup>1</sup> the molecular concentration of sweat is decidedly under that of blood serum it is always a question whether by this procedure an absolute substitute of the renal function in regard to the removal of urinary substances is attained. On the contrary, it might be questioned whether the mere withdrawal of water would not rather tend to increase the concentration of the blood serum, producing an unfavorable action, in that uremic symptoms would be more rapidly produced. In fact, every experienced physician has seen one or the other example of the appearance of uremic phenomena soon

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<sup>1</sup> *Fortschritte der Medicin*, Bd. xix, Nr. 21.

after a hot bath or a hot pack. But these are for the most part rare exceptions, whereas usually a subjective and objective amelioration follows the procedure. However, the experience just described makes it necessary to watch the patient after this sweating process has been attempted, in order to note the momentary success. Perhaps the undoubted frequent use of this measure does not consist in the withdrawal of water but in the regularly marked flow of blood to the skin, in which way the main object is attained.

The value of depriving the renal circulation of blood by *direct withdrawal*, with an increased experience, has become more and more unquestionable to me, so that I should like to advise this method of treatment rather than that in which sweating is produced. As soon as, with a decline in the excretion of the urine (immaterial whether this contains much or little blood), the first symptoms of intoxication occur, i. e., headache, vomiting, I lose no time but apply to each renal region, according to the age of the child, one or two leeches. The succeeding bleeding is to be kept up for a little time afterward. Sometimes I have seen that it has been rather severe and continuous so that compression or other measures were necessary to stop the hemorrhage. However, it is better when a little too much than too little blood flows. The leech itself withdraws about 10 grams of blood and if (with 2 leeches), therefore, a similar quantity, 15 to 20 grams, flows from each wound, this is of advantage. Soon afterward, in favorable cases, the secretion of urine rises again, and headache and nausea disappear. In the severer cases, however, the improvement is not sufficient, it is transitory, and headache returns. Cloudiness of the sensorium and a convulsive attack are added. Then without more ado there should be a general blood-letting by means of *venesection*, by which 100, 150, or even 200 grams of blood in older children, may be withdrawn. I have gathered quite a number of observations in my clinic as well as in my consultation practice in the last two years, in which venesection has been followed by life-saving success. This may be followed by a subcutaneous injection of a normal salt solution of 100 to 150 grams directly. The secretion of urine is permanently increased; it is still bloody for some days, but is copious; the convulsions do not return and the beginning amaurosis gradually disappears.

If in a severe scarlatinal nephritis the kidney is examined then, with the extended stasis of the blood in the glomeruli, in the cortical capillaries, in the vasa recta, the connection is not very difficult to understand. Without increase of blood pressure, by means of the general diminution of the amount of blood, the renal blood channels can be more readily traversed. Besides, perhaps the removal of a part of the retained substances of the blood serves a further purpose. It may therefore be a condition which allows the organism to return to a normal state. A certain advantage is attained by the use of warm poultices in the renal region.

After the danger of uremia is over, or at the same time with it, it is necessary to combat the cardiac asthenia in severe cases. Even the subcutaneous salt infusion is of value here. But the question arises, whether drugs should be used. Here, with rapidly approaching danger, camphor, even in repeated

large doses, is valuable, but more frequently instead of combating a rapid collapse we must treat a permanent and slowly advancing asthenia. Regarding the favorite method of treatment by digitalis, at least so long as the case is in the acute stage, I have an aversion, which is perhaps not justifiable, but which is based upon a momentary increase in injury and a fatal ending in a previous severe case of nephritis. Care in the employment of this potent remedy is necessary under all circumstances.

Steffen advised in these cases of cardiac asthenia to administer ergot in fair-sized doses, three to four times daily. I followed his advice and employed the fluid extract. In fact it appeared to me to be occasionally useful in combating the dilatation which gave rise to cardiac weakness.

Occasionally the subcutaneous injection of *strychnia nitrate* once daily has a beneficial influence upon the cardiac activity.

*Caffein* should also be considered, as it shows a simultaneous action, as a stimulant for the weakened heart muscle and as a vasomotor remedy. Naturally, also on account of the simultaneous influence on the kidney, the remedy must be given guardedly.

Then by plentiful nourishment the action of the heart may also be restored. According to the investigations of v. Noorden, no great fear need be attached to the administration of other albumins than of the casein of milk, as was thought to be the case formerly. It is best in nephritic cardiac weakness to arrange the diet so that, with moderate quantities of albumin, carbohydrates are also mingled with the food. The treatment of these conditions requires the greatest care as well in the choice of a drug as in a nutriment.

[As the result of a long and various experience I join in the author's warning against the use of digitalis, mainly in acute cases of nephritis. Besides, on account of the frequency of vomiting, the drug should not be given because it causes vomiting. Its taste is bad, besides. Strychnin nitras or sulphas may be injected in gr.  $\frac{1}{80}$  to gr.  $\frac{1}{30}$  several times a day. The best salt of caffein is the salicylate or benzoate of sodio-caffein. As this double salt is soluble in two parts of water it is conveniently given hypodermically. Internally 1 grain which contains  $\frac{1}{2}$  gr. of caffein, may be given every hour or two hours; more in older children. A small radial pulse indicates the use of nitrites. The sodium nitrite may be given internally in doses—daily—of 3 to 5 grains, the trinitrin (nitroglycerin)  $\frac{1}{100}$  gr.—more or less—daily, in many broken doses.—EDITOR.]



## DIPHThERIA AND DIPThThERITIC CROUP

By A. BAGINSKY, BERLIN

DIPHThERIA is a *contagious* disease, directly transmissible from child to child, also indirectly from a third person and infected fomites. It is one of the most terrible scourges, and has appeared in decimating epidemics which are scarcely equalled by any other affection in the history of medicine. It is not our purpose at this point to report them, and we must refer to special publications regarding this point. From these it can be seen that the first epidemics go far back into ancient times, that the disease bore the name "Morbus ægyptiacus," and "syriacus," that on account of its terrible character Spanish authors gave it the name "*Garotillo*" (so-called after the cudgel of the executioner)—and, on the other hand, by Swedish physicians it was given the name "*Strypsjuka*" (strangling disease). From these designations, which were popular with the people, it may be seen that the danger of suffocation was the most prominent symptom of those affected, and that death by suffocation was the most feared result. And yet so many other characteristics were found in the ancient descriptions of the disease: The rapid prostration, the decline in the cardiac power, the condition of delirium, etc., jactitations, effusions of blood, etc., which all point to the fact that still another element other than the purely mechanical hindrance in respiration was included in the disease, and showed itself prominently by causing death. And this was perhaps the reason that, according to the mode of its appearance in one or the other form, two different diseases were distinguished, and the actual suffocating disease which was later designated as "croup" by the Scotch, was differentiated on the other hand from that form which apparently caused death by *poisoning*. The clinical picture of the disease varied in spite of the plentiful opportunity for its observation, up to that time in which the great French physician, Pierre Bretonneau, combined the whole, and from a number of classical observations that could not be surpassed, called attention to the *unity of the various forms*, in that he showed the combination of the appearance of thick leather-like membranes in the mouth, pharynx and naso-pharyngeal space, and with the changes in these structures, malignant and deleterious symptoms at the same time were present. Bretonneau had attached the greatest importance to these membrane-like pathological formations, and also originated the name diphtheria (from *διφθέρα*, the skin), which the disease has retained.

After these brief remarks regarding the name of the disease, we shall at once consider the clinical course and observe the manifold pathologic pictures which are combined in this conception of a substantive disease which shows so many anomalies. Unfortunately, the number of cases in every large hospital that treats this disease is so great and varying that it is possible to demonstrate almost everything which is designated as belonging to the diphtheritic process, even all the sequels and complications of the disease.

*Case History.*—Boy aged three years, showing some pallor, apparently well nourished, with a moderately high temperature ( $101.3^{\circ}$  F. to  $102.6^{\circ}$  F.), admitted to the hospital yesterday. The history did not show accurately the time at which the boy was taken sick, but the onset of the disease could not have been long ago as the little patient appeared to be well for a few days previous, ate and played. It can be heard from his crying that he has a clear voice, nevertheless, the tone is somewhat changed, it may be expressed as pharyngeal. Upon observing the throat externally, it appears to be somewhat thickened; upon palpation two glands at the angle of the jaw are enlarged almost to the size of walnuts; apparently they are painful. The skin of the patient shows a normal color, is not especially hot nor dry, nor is there marked perspiration. The physical examination of the thorax and abdomen shows nothing abnormal. The pulse is soft, but little tenseness in the radial artery. The pulse rate, upon which little importance must be placed in children, as it varies greatly under slight emotional conditions, is somewhat over 100 per minute.

Upon examination of the mouth and *pharynx* it will be noted that the lips are dry, slightly covered with sordes; the tongue is red at the margins, white upon its upper surface. Upon opening the mouth wider, there are noted upon both tonsils and posteriorly on the wall of the pharynx a *thick, grayish-white coating*, arising from the surrounding tissues, apparently tightly adhering to the surface. The mucous membrane which has remained free is of a pale red color, not too darkly tinged, therefore, not specially inflamed, still it is swollen, perhaps slightly transparent as if moistened through and through.

This corresponds with the classical description given by Bretonneau—"Diphtheria, a membranous coating which covers the pharynx."—The child has diphtheria.

Before answering the question, "How has this disease originated, and to what symptoms does diphtheria give rise?" let us attempt to form a picture of the course of this, apparently, by no means terrible affection.

This can be seen from a *second case history*. Child about three years old, has been under treatment for a few days; it showed the same symptoms as the former case when admitted, but at a glance it can be seen that it is of an entirely different, much less severe type. Yet, if we did not know that a few days ago it was seriously ill, we should scarcely take it for a sick child on account of its joyful, happy disposition. The temperature curve of the child shows that the temperature, which was originally somewhat over  $102.2^{\circ}$  F., has gradually fallen to between  $98.2^{\circ}$  F. and  $99.5^{\circ}$  F. The pulse is soft, still over 100 per minute. There is no impediment to the respiration; the voice, as may be noted when the child speaks, is pure and the physical examination of the respiratory organs shows nothing abnormal. The two submaxillary lymph glands at the angle of the jaw, that can scarcely now be felt, are but slightly conspicuous. The lips are smooth, of a pale red color. The tongue is still slightly coated. The pharyngeal mucous membrane appears upon the whole to be of a somewhat deeper red than normal. The tonsils are larger than normal and appear dark red, and upon close examination we still note here and there individual grayish-white streaks lying upon them, whereas the rest of the pharyngeal membrane appears red. Upon the posterior wall of the pharynx a broad thread of mucus of a yellowish-gray consistence

is noted. This is all that remains of the original grayish-white, thick membrane. The examination of the urine shows that there is nothing abnormal, especially no albumin is present. This has all occurred *under the influence of an effective therapy*, the diseased process having given way to an almost normal condition, and we are entitled to hope that nothing will occur which will prevent the speedy return of the entire process to complete health.

Upon the basis of these two clinical histories, we shall enter upon a general discussion of the affection.

*Diphtheria* is a disease that is communicable; it usually begins with fever and commonly arises from the pharyngeal organs or often from the nasal mucous membrane. It is characterized by pseudo-membranous yellowish-gray or dirty gray deposits which are adherent and invade the mucous membranes, showing a tendency to distribute themselves. The disease may appear in the mildest form, although with some little fever, with but relatively slight implications of the general organism, and so appear and run its course as a *local disease of the pharyngeal structures*.

The disease is not limited to *climatic or geographical locations*, it has appeared in the coldest as well as in tropical and subtropical regions, and everywhere has shown an epidemic distribution. Nevertheless, we cannot suppress the impression that the affection shows a preference for the temperate and northerly zones; at least it shows itself there more frequently than in others, and its epidemics are of longer duration and perhaps also more severe. The highly situated portions of the different localities show no exemption from the appearance and distribution of the disease, neither does the constitution of the ground have an influence; the inhabitants of swampy areas are affected to the same extent as those of the mountain regions, the inhabitants of sandy and gravel districts as well as those dwelling upon more compact earth.

The question has been much debated whether *season* favors the distribution of diphtheria. Unquestionably the months of the winter are those in which the affection appears most frequently and most severely, and in which it attains its greatest distribution; still there is frequently enough an opportunity of observing the severest epidemics in summer, and I can still recollect with horror the first epidemics that I saw in the summer among the population of the country. However, it is possible that the affections of autumn and winter, particularly the catarrhal implication of the pharyngeal mucous membrane, produces a predisposition to the disease.

The affection scarcely ever manifests a preference between rich and poor, and affects those living under the best hygienic conditions, even the members of royal families having succumbed to the malady. Naturally, the unfavorable mode of life of the poor, the bad housing, crowding together, and the uncleanness which goes hand in hand with these, favor the distribution of the disease. However, it cannot be proven with certainty what was formerly generally accepted, that bad ventilation of rooms alone, or faults in flow of dirty water and gases from privies, favor the distribution of the disease.

The affection occurs at any *age* and is widely distributed; the cases are



by no means rare in which parents are simultaneously attacked with their children, and in which the disease is frequently communicated to physicians from their little patients. Regardless of all this, the disease specially seeks the youthful and is most dangerous in them. According to our own observations, children in the first year of life are but seldom affected, but as early as in the second year quite large numbers are attacked; a maximum morbidity and also mortality figure is shown by the period from the second to the fifth year of life. This corresponds also to the observations of other investigators. They have not been able to determine that *sex* shows any difference in the predisposition to the disease, on the contrary, boys and girls are equally affected. Neither do different *constitutions* show a variation in predisposition; but one point must be observed, that especially such children as suffer from chronic catarrh of the nasopharyngeal space, from enlarged tonsils and adenoid vegetations are attacked; also certain *pre-existing affections* produce an increased *predisposition*, such as measles, and, above all chronic diseases, such as poliomyelitic paralyses, hemiplegias, etc.

On the other hand, the very remarkable fact has been discovered that there are persons that are actually immune to the disease; and it is probable that this *immunity* is due to a peculiar constitution of the blood serum.

As has already been emphasized, the disease is *contagious from person to person*. The pathogenic organism, however, also adheres to fomites, such as clothes, toys, and may be transferred by means of them. But the transmission by this means occurs to a much slighter extent than by direct contact. Curiously, in spite of the daily experience regarding this contact-infection, there are some physicians who doubt this property, and just such instances have occurred in which physicians were infected, and these are more convincing than any others. Even Bretonneau mentions cases of this kind with a fatal outcome, and any physician of experience is able to relate some very dangerous examples of that period when it was not yet possible to take early and decisive measures against the disease. If in our practical activity, attention is paid to these points, we will note daily examples of transmission among children of the same family, school children, and from servants and nurses to children of the family. The most unpleasant and even momentous circumstance is this, that adults who do not appear to be seriously ill and only suffer from mild forms of angina may transmit the most severe forms of the disease to children. For example, some years ago my own child was infected by a servant who I ascertained afterward had visited her sister affected with diphtheria, the servant in question suffering from an angina which she had concealed from me. The cases are well known in which the further dissemination of the disease has been promoted by children affected with diphtheritic disease attending school. As slight as the transmission by fomites may be compared to the *transmission by direct contact*, this must, nevertheless, be admitted. We may assume as quite certain that transmission occurs by means of infected food products, milk, etc., although it may be difficult, in the individual case, to determine this mode of distribution of the disease.

How this transmission and dissemination occurs will be readily understood when we come to describe the cause of the disease, **the bacillus of diphtheria**. It is well known that after much fruitless and discouraging labor, after prolonged search, Löffler, in 1884, succeeded in cultivating a bacillus from the membranes of diphtheria, which had been previously noted by Klebs and described by him as the likely cause of the disease, but which still lacked an invariable demonstration, and for this reason was not fully proven. Löffler described the bacillus as a *constant finding* in the dense pseudo-membrane of typical cases of diphtheria; in small heaps below the surface of the pseudo-membranes where the bacilli are present in large numbers they take an intense stain with the Löffler color-mixture. Löffler also gave the elective mode of cultivation upon a blood serum mixture, which since then has shown itself to be the best culture medium for the bacillus. The mixture consists of 3 parts of calf's or lamb's serum, 1 part of neutralized veal bouillon, 1 per cent. of peptone, 1 per cent. of grape sugar, and  $\frac{1}{2}$  per cent. of table salt. The bacillus shows itself as a club-shaped organism  $1\frac{1}{2}$  to 2.0 microns long and 0.3 to 0.5 microns thick, usually as double rods so situated that they are joined at an angle (compare the illustrations, Figs. 33 and 34). In pure culture the microbes present a special appearance; the latter is somewhat characteristic in that the rods for the most part are parallel to one another, showing a palisade arrangement.

I cannot enter here upon the other special peculiarities of the bacillus, nor upon the many and manifold doubts which were thrown upon it, especially after a bacillus was discovered that resembled it, which was finally designated as the *pseudo-diphtheria bacillus*, which is, however, non-virulent, whereas the utmost virulence is a peculiarity of the diphtheria bacillus, and was proven in the most certain and convincing manner by Löffler in animal experiments. The diphtheria bacillus grows luxuriantly upon the slanting Löffler serum, in a few hours becoming microscopically visible, showing a pale, yellowish-green, vapor-like streak which appears finely granular upon the surface, or in wavy forms, with small roundish furrows upon the borders.

What appeared to oppose Löffler's important findings was primarily the fact, that succeeding investigators did not always find the bacillus in all cases diagnosed as diphtheria, until finally Roux and Yersin in Paris demonstrated its presence in methodical examination, first in 61 certain cases of diphtheria, whereas in 19 others which originally had the appearance of diphtheria, but in their further course showed themselves as non-diphtheritic, it was absent. Simultaneously with the French authors, we began our own investigations in this hospital and we succeeded in demonstrating that the Löffler bacillus was always present in cases which showed a severe course, whereas those cases that were looked upon as diphtheria, but in which the bacillus was not present, showed an entirely different, i. e., more favorable course, a fact which was proven by all our further investigations. Hence we could say with absolute certainty that the Löffler bacillus, in the cases



admitted to this hospital in which a diagnosis of diphtheria had been made according to the clinical findings, was absent in scarcely 3 per cent., and in those cases in which upon repeated careful examinations the Löffler bacillus was not found, with but very isolated exceptions the clinical course showed that they were *not cases* of diphtheria. This assured the significance of the bacillus as the pathogenic agent of diphtheria and not the slightest contradiction to this fact can be advanced, even to the present time, for the bacillus is found upon mucous membranes upon which no pseudo-membranes are present, and in which no actual symptoms of disease can be demonstrated. This latter fact, on the contrary, is connected with the immunity of the culture media in individual human beings, and no less so with the greatly varying virulence of the bacillus, which like all pathogenic bacteria without exception is very unstable and varies greatly; this, however, does not in any way alter the clinical facts that have been determined and they must be adhered to.

I may assume that it is well known that the bacillus shows certain properties which explain the manner of the distribution of the disease such as its extraordinary tenacity of life, how it flourishes in milk, how it adheres to fomites, such as under-clothing, boots, clothing, etc., further, its property of growth in association with streptococci, bacillus coli, bacillus proteus, etc., in which it has even been shown that some of the previously mentioned organisms are occasionally capable of increasing the symbiotic growth of the virulence of Löffler's bacillus, or to rekindle it. On the one hand, this explains the transmission of the disease by fomites, of which we have already spoken; on the other hand, the upflaring of severe epidemics at one point and the milder course of the disease at another point. Let us consider for a moment the mode of action of the bacillus in the affected organism. Obviously, the pathogenic agent changes into a morbid condition the affected mucous membrane at the point of its attack, which process may be recognized in the appearance of the diphtheritic pseudo-membrane. What is this pseudo-membrane anatomically, and how does it arise?

The anatomical changes in the mucous membranes which are attacked by the pathogenic agent and the pseudo-membranous formations have been for a long time the objects of closest observation, without complete unanimity of opinion having been attained even to-day. We cannot repeat in detail the various differing opinions, and I may refer, as I believe, to my rather exhaustive compilation in my book upon diphtheria and croup.<sup>1</sup> I can only repeat here that it is due to a high grade of degeneration of the vessels and fibrinoid degeneration of the connective tissue, causing a fibrinous exudation, the products of which, upon the surface, are subject to necrosis and absorption, becoming a granular mass; with all this there is a dissemination of the entire new-formed mass, of the bacillus and other more accidental microbes (streptococci, staphylococci, bacillus coli, etc.). Therefore, in a combination of

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<sup>1</sup> A. Baginsky. "Diphtherie und diphtheritischer Croup." Alfred Hölder, Wien, 1898.



necrosis with exudative fibrinous desquamation, what is especially conspicuous is the extraordinarily great tendency to the propagation of the process, so that the membranes, as already described by Bretonneau, appear to "flow downward," although they are tightly adherent and the mucous membrane is altered to some distance below the surface.

The distribution of the process to the larynx, trachea and bronchi causes deposits in the respiratory tracts, producing suffocative phenomena and threatening death by choking. From the mechanical distribution of the process, however, the other severe symptoms and fatal cases that have nothing to do with suffocation cannot be explained. Here the second factor comes into play, that a severe active poison is produced at the point of action, entering the lymph and blood channels by osmosis and being conveyed by the blood and lymph streams to distant organs and tissues. Löffler in his first studies arrived at the opinion that the bacillus found by him produced a poison, and with this he explained the fact that the bacterium brought about intense general action in the affected or artificially affected organism. He also succeeded in introducing the poison from precipitated glycerin extract of bouillon cultures by alcohol and he was able up to a certain degree to determine experimentally its deleterious effects. The French authors, Roux and Yersin, went further, they discovered that the toxins elaborated from Löffler's bacterium brought about phenomena in animals which were throughout analogous to the severe diphtheritic symptoms in human beings; for example, certain forms of paralysis may be mentioned, the consideration of which we shall enter upon clinically somewhat more minutely. Regarding the nature of this toxin, its probable chemical constitution, its terrible action on the animal organism, these cannot be discussed in detail here; it is sufficient to call attention to the fact that it has been carefully studied, and that especially from these studies the wonderful knowledge of the antitoxic action upon the animal organism developed, which Behring utilized therapeutically in determining the antibodies which were secured from the blood.

Upon the basis of the conclusions which we have reached, it is found that *a priori* the diphtheritic affections may be divided into two principal groups, first into that group of milder affections in which the author has been successful in limiting the area of the disease and rendering harmless the toxins produced at this point; these are the purely local diphtheritic affections, in which the general action of the poison does not occur. And then the second group, the general constitutional affections, in which the local limitation of the process is not successful, and the further distribution from the point of the first attack is rapid and marked, in which, above all, by the entrance of the toxins into the lymph and blood mass, severe and irreparable disturbances occur in distant organs.

*Limited, local* diphtheria will always represent the mild, and *general diphtheritic intoxication*, with implication of distant organs, will include severe forms of the disease. By the term **diphtheritis**, the local limitation of the process is meant, by the term **diphtheria** the general involvement of the

organism is characterized, and we shall conform to this terminology, which I believe is very proper.

Let us return to the histories of the two cases just quoted. It is not difficult to determine that in both cases we are dealing with forms of local diphtheritis.

The further distribution of the process in the first patient is hardly likely, after the therapeutically protective prohibitive measures used by us; in the other case it may be excluded with certainty. We may expect that in the first case the membranes which are still present will soon loosen, will melt away, similar to the condition which has already occurred in the second case, without any deep lesions, ulcer formation or similar conditions remaining, also without general phenomena appearing later. In fact, these tissues are restored to complete health after a relatively short time, in from three to four to six days. Any one who has the fortune to observe only such cases of diphtheritic disease will hardly believe that the affection with which he is dealing belongs to the most terrible scourges of the human race.

Is there then in any case of a locally appearing diphtheria from the onset the certainty of the local limitation of the process, and with this the fortunate undisturbed course of the disease? As a rule, by no means! During the time, which is now fortunately of the past, in which we were unable to make our patients "toxin-resisting," and to immunize them in time against the local effects of the poison, we were certain of no case, neither from the later propagation of the process to larynx and trachea, nor from the danger of suffocative phenomena, nor from the appearance of severe later lesions, in particular severe cardiac phenomena, nephritis, and various forms of paralysis which often arose several days after complete sloughing of the membranes from pharynx and tonsils. For this reason the prognosis of the disease was very questionable, even in the apparently mild local forms, and this was true to a greater degree the longer the process continued, and the membranes remained upon the pharynx and tonsils. This has now fortunately become different, since it is possible to immunize the body with the serum of diphtheria if we use it in time and in sufficient quantity. Later, in describing the serum therapy we shall return to this consideration. However, at this place it must not be omitted that even without the action of serum, forms of the disease remaining local have occurred in every, even in very severe epidemics. Here the affected organism was either able itself to produce protective bodies in sufficient amounts or it chanced that the microbes that brought about the local affection were not sufficiently virulent, so that this acute action of the toxins did not occur. However, I should advise not to depend upon such fortunate happenings in any single case; it is well to remember that no case, even though apparently mild, should be trusted.

For this reason an active therapy is necessary in every case, no matter how mild. Generally in these local forms of disease a local therapy will be thought of. This is certainly not unjustified, and we shall learn to recognize a number of remedies which have the effect of combating the local lesion,

that are commonly used and are by no means absolutely ineffective, even if they come far short of bringing about what was theoretically expected of them. We could here speak of these therapeutic effects, but I believe it is better to first describe general varieties of the diphtheritic disease and only then to speak of all therapeutic measures in connection.

After having described the mildest local form of the disease, let us turn to the most severe, the **septicemic general affection**. This may be designated as a special division of diphtheria, and I shall describe a case from my former experiences, which, unfortunately, was rich enough in this respect. As a matter of fact, this is a terrible form of the disease, a form which has caused diphtheria to be the terror of entire nations, the specter of annihilation of entire families. Picture to yourself a waxy pale, swollen face, with a hollow glance, semicomatose, with open mouth, shrinking position of the body, the head deeply sunk into a pillow. The nose appears thickened, the nasal openings sore, and a yellowish irritating secretion is slowly seen oozing forth, which irritates the upper lip making it sore, red and thickened. The lips are dry, bloody, fissured. The entire anterior part of the throat appears unevenly thickened, and upon palpation the cervical lymph glands are felt to be enlarged to thick structures, which are not uniformly limited but changed into a general doughy swelling. The child snores, the eyes are half opened, the conjunctivæ are visible, slightly injected, and the angles of the eyes usually somewhat purulent. A frightful fetor is noted in the respired air and almost forces us to remain away from the child. The tongue is red, dry, the papillæ standing out prominently. The examination of the mouth, the tongue and the pharynx causes bleeding, which not infrequently is copious and difficult to control. The entire pharyngeal region presents an appearance which is actually terrible. Greenish-gray to black hemorrhagic masses clothe the pharynx, and nothing more is visible of a normal mucous membrane; the normal pharyngeal structures cannot be recognized. The velum palatinum is markedly thickened, and arising from it, reaching to both sides and anteriorly over the entire hard palate are seen thick, flattened, grayish-black pseudomembranes. Usually the voice of the child is hoarse; the cough which is due to the examination is hoarse, barking; the respiration is somewhat difficult, and, upon inspiration and expiration, slightly impeded without a conspicuous dyspnea being present on this account.

*The constitutional symptoms* correspond to this terrible picture. Occasionally here and there upon the cutaneous surface, distributed petechiæ are found, or deeper-formed striæ, hemorrhages which are distinguished from the surrounding intensely pale areas by a dark bluish-gray color. The pulse is feeble, can scarcely be felt at all, or is even absent; as a rule, however, it is accelerated, at the same time irregular; the impulse is weak, the heart sounds dull; the belly is edematous; spleen and liver are enlarged; the urine mostly contains albumin; the bowels are constipated, and if a movement is brought about artificially it is of a very dark color and very offensive. The temperature is but slightly raised, scarcely over 100.4° F., occasionally even sub-



normal. This is the condition that, as a rule, after two or three days leads to death. In the rare cases in which these septic forms of the disease terminate in recovery, this occurs by ulcerative sloughing of the gangrenous masses and a gradual cleansing of the many deep ulcers produced by this and not without severe general phenomena on the part of the heart, the kidneys and the nervous system, which we shall describe more in detail later on. Weeks and months may pass before such patients recover, and in some cases a persistent damage to the heart remains, which may still be noticeable for years and bring about the early death of the child.

Let us examine the anatomical changes which are met with in autopsy of these severe cases. The pharyngeal structures show the picture of severe gangrene; deep into the mucous membrane a grayish-black coagulated and decomposed mass forces its way; the tonsils have almost disappeared, the deformed structures of the pharynx are scarcely recognizable; and over the thickened, tumorous epiglottis the same smeary grayish-black or greenish masses enter the larynx, leaving but portions of the true and false vocal cords recognizable. Below the arytenoid cartilage and the cricoid cartilage, looking into the trachea, over the dark red, here and there hemorrhagic mucous membrane, fine fatty structures representing thin membranes are noted, which fluctuate under a stream of water. The lungs are hyperemic, partly atelectatic, partly filled with bronchopneumonic infiltrates. The liver is large and soft, the parenchyma is cloudy; the spleen is also large, cloudy, less consistent, of a grayish, dull appearance, and upon the cut surface the follicles are hardly recognizable. In some cases special changes are shown by the stomach; the swollen and thickened mucous membranes show distributed, deep, dark brownish, hemorrhagic deposits in striæ form, radiating from the cardia, and grayish-yellow deposits are noted in the centre of these striæ, which deeply penetrate into the hemorrhagic areas; occasionally the same changes are noted in the intestinal canal; as a usual thing Peyer's plaques are enlarged as in enteric fever, stand out prominently, are injected and even here and there hemorrhagic, or even ulcerated. An ugly gray cloudy appearance is noted in the musculature of the entire flaccid and dilated heart. There are also cases in which fresh coagula, which are adherent, apparently having arisen during life, can still be noted between the trabeculæ of the auricle or of the ventricle. All in all a typical picture of septic changes. The bacteriological examination of the fresh organs at autopsy allows us to cultivate the Löffler bacillus usually in combination with the streptococcus, whereas from the necrotic portions of the pharynx a rare mixture of bacteria is grown, among which the Löffler bacillus is naturally predominant. We see, therefore, in this form of disease, besides the terrible action of the toxins of the bacillus in the region of its primary attack such as pharynx and nose, the phenomena of the general action of the virus in all organs, which may be recognized as the usual process, damaging the tissues in a most deleterious manner.

The question has been much discussed, whether in the cases described,

the Löffler bacillus is the actual cause of these terrible changes or whether this action may be attributed to other microbes. The symbiosis with dangerous streptococci is certainly calculated to increase the virulence of the bacillus, nevertheless, there are enough cases to be observed in which in these changes the Löffler bacillus occurs in almost pure culture, so that, without doubt the most malignant lesions may be due to it alone. For this reason the designation of these malignant septic forms as *Diphthérie à Streptococque*, which appears to be a favorite with French physicians, is not quite correct, it does not even require the presence of streptococci to bring about even the severest pathological phenomena; a high grade of virulence of Löffler's bacillus is alone sufficient, and for this reason this microorganism dominates this as well as the previously described pathological picture.

Between this terrible affection and the previously described mild local forms, lie the other cases of *diphtheritic general affection*. Naturally, with serum therapy, as has already been mentioned, the clinical picture and course of the disease has changed so much that we only meet with these severe forms in cases that come under treatment very late, in which the virulence of the bacillus has been able to attain its full action. It will be well to look at the clinical history of some cases to illustrate this.

Girl aged ten, taken ill eight days ago, showed lassitude, anorexia, and symptoms of general malaise. Four days ago she began to complain of throat pains, enlargement of glands noted on both sides of the neck; the child began to have a fetid breath, and evidently had high fever. Admitted to the hospital without having been treated by a physician. On admission, the first glance shows that we are dealing with a severely ill child. The face is pale, somewhat puffy. The sub-maxillary region swollen upon both sides. The glance is feeble, the entire musculature flaccid, the mind is clear; the voice is not hoarse, but only sounds suppressed, pharyngeal; the lips are dry, fissured. The tip of the tongue is dry, the superficial surface is covered with a grayish coating; the mucous membrane of the mouth is generally dry, only tough, pappy mucus adheres between the tongue and the palate. A serious picture is presented by the pharyngeal region. Both tonsils, the velum of the palate and the uvula are transformed into a greenish-gray thick pseudo-membranous mass. The uvula hangs like a rigid lump of a greenish-gray color upon the dorsum of the tongue; the enlarged tonsils almost meet in the median line, so that it is easy to understand the suppressed tone of the voice and the somewhat prolonged pharyngeal inspiratory murmur which is present. The gray membranous masses reach almost to the middle of the hard palate. The respiration is not actually hindered, but still it is irregular, about 20 to 25 respirations per minute. The pulse is bad, the wave low, the tension slight, 120 per minute. The cardiac sounds are very weak, dull; the size of the heart is normal in so far as can be ascertained by percussion, although the heart appears to be covered by the pulmonary borders. The temperature is 102.2° F. The urine contains much albumin; it also contains morphotic elements, cellular, somewhat decomposed substances and parts of casts. The rest of the organs show nothing abnormal.

This represents one of the severe cases of *general diphtheritic infection*.

Now let us observe the course which the disease ran in such cases, as a rule, during the period when we were not as yet in possession of a specific remedy, and which it may even pursue to-day if the remedy is only applied eight days after the beginning of the case, and its favorable action is not attained. Mostly, or at least in many such cases, the process which was present in the pharynx rapidly led to gangrene and the complete picture of sepsis developed, which we have described previously. Then the children sank more and more into a comatose condition and succumbed under the



phenomena of adynamia and cardiac collapse. With this the fever need not rise to high temperature ranges, nor need special complications develop. Or, and this is the better or the only course of the disease, the children began to improve, the pulse became better, and the albuminuria diminished; simultaneously, a reactive inflammation occurred in the pharynx, which prevented the membranes from spreading and produced desquamation at the margins of the pseudo-membranes. This gradually caused the membranes to loosen by disintegration, and after three to four to six days it could be noted that the pharynx had become free, the swelling had diminished, the red mucous membrane of the velum and the tonsils again appearing. With this, the entire condition of the child had improved; the lips no longer were so fissured, the tongue no longer so dry as formerly, the fever had declined. Convalescence began and uninterruptedly ran its course.

However, this was the rarer case. More frequently, only during this time, those threatening and dangerous morbid phenomena occurred which pointed to severe damage to internal organs, the heart, the kidneys and the nervous system. What characterizes the course of these diseases now since we have employed serum treatment is this, that what was previously the exception is now the rule, that, usually, with the desquamation of the pharyngeal coatings, the decrease of the swelling of the mucous membrane occurs, and with it complete euphoria. Naturally, only then if not too long a period intervenes between the onset of the disease and the employment of the remedy.

Still a third course is open to these cases, i. e., the further distribution of the pseudo-membranous masses to the larynx and trachea, and with severe suffocation threatening life, the phenomena of *diphtheritic croup* appear, which require prompt surgical interference.

All of these methods of termination we shall now have to study. They may be studied in those cases which, although treated with curative serum, did not remain free from *complications*, because the employment of the remedy was too late or it was used in insufficient amounts.

This can be studied from case histories in which **diphtheritic cardiac symptoms** are very characteristically shown as the expression of a *lesion of the heart*.

Girl aged four years; upon the fourth day of her disease became markedly worse and was brought to the hospital. The child was well nourished but was cyanotic from the onset. The voice was clear, not hoarse, dyspnea was not present. The pulse was small and easily compressible, 138 beats per minute, the temperature was 101.8° F. Small quantities of a mucoid secretion flowed from the nose. The pharynx was markedly swollen, the vault of the pharynx, uvula, tonsils and posterior pharyngeal wall were completely covered by a smeary, gray, quite thick, pseudo-membranous mass; there was fœtor ex ore that was quite unpleasant. The heart showed nothing abnormal; especially were the heart sounds clear. Lungs normal. The urine was of acid reaction, contained moderate quantities of albumin, and showed, among morphotic constituents, swollen and partly degenerated, granular as well as epithelial cells, containing fat granules, also hyaline casts covered by cells and granules. The further course of the affection was completely free from fever, the membranes loosened slowly. During the entire time, in spite of the apparent euphoria, it was conspicuous that a certain coolness of the extremities was present, with a small, easily compressible pulse, varying between 118 and 140 beats per minute. Upon the eleventh day of the disease a slight paralysis of the velum of the palate was noticed, and, apart from the somewhat nasal voice which still existed for some time, was easily recognized by the fact that fluids that were taken



easily caused paroxysms of coughing and choking and were also regurgitated through the nose. In the meantime the heart-sounds had become peculiarly dull, and it was noticed in the radial pulse that an intermission of some beats occurred from time to time, this giving the pulse a certain degree of arrhythmia. Up to this time the albumin had not entirely disappeared from the urine. If the heart in this stage is examined it will be noticed that the impulse is weak, scarcely to be felt, percussion of the borders of the heart is not changed, but auscultation shows a peculiar character of the heart sounds which has been designated galloping rhythm. This is a peculiar beat of the heart showing three beats, not always with the same accentuation of the sounds, usually so that the principal accent occurs upon the first beat, but also that the second among the three beats, therefore, the middle tone, is principally accentuated. This peculiar rhythm appears to arise in that the ventricular contraction occurs in two different periods; it is especially then a certain sign of severe disturbance of cardiac action and weakness of the heart muscle if it is not quite rhythmical. This phenomenon is always very important and requires the greatest attention, as it not rarely indicates beginning destruction of the heart muscle, perhaps also of the *cardiac ganglia*, and is thus the first sign of a beginning fatal issue due to the heart. Fortunately, this is not always the case, and in the history just quoted it is possible that the little patient will recover and go on to complete convalescence.

However, we must not conceal the fact that the condition is very dangerous, especially if we recollect the sad results which under similar conditions have occurred too frequently. Under like circumstances, as in the case just quoted, cardiac death has occurred, it being impossible to prevent this issue. It may take place in two different ways: In the midst of apparent euphoria, suddenly vomiting may occur and a very serious condition of collapse take place, which rapidly and quite suddenly leads to death; then the pulse disappears suddenly from the radial artery, the child becomes remarkably cyanotic, and later on pale. The extremities, the tip of the nose, are cold; the respiration irregular and may become stertorous, and in spite of all analeptics, the child dies in the arms of the nurse. Or death due to the heart may occur slowly, but is just as certain, in that varying collapse-like conditions and the adynamic phenomena finally increase. In the latter case paralyzes are usually present and the kidneys are affected; with vomiting, difficulty in respiration, severe abdominal pains occur. The lips are pale and cyanotic. The extremities are cold, the pulse can scarcely be felt, thready. The cardiac impulse is weak, minimal, tachycardia is present, so that up to 200 rapid and irregular contractions occur in a minute; or the cardiac contraction becomes increasingly more sluggish and slower, 36 to 40 beats in a minute. Vomiting occurs more frequently, briefer pauses of improvement intervening, which give fresh hope. However, the collapse phenomena return, simultaneously the liver enlarges, becoming larger and larger, and may be palpated as a large stone-hard tumor, reaching down into the pelvis. The child has become apathetic to the highest degree, the eyes have a glassy look, the tongue and the tip of the nose have become cold; respiration is weak, scarcely perceptible. Tired, exhausted almost to the point of death, sometimes with a completely clear mind, it turns upon the side and gently slumbers without producing a sound, never to awaken again. Involuntarily, even if it were not known, the physician and all those surrounding the patient recognize the action of a terrible poison, which apparently has shown its action by paralyzing the heart muscle.

If we ask ourselves what anatomical changes the heart muscle in these conditions and processes has suffered, we would be greatly mistaken if we assumed that they are always very marked or constantly of the same nature. Occasionally, in fact, nothing is found which could at all explain the loss in the action of the heart, and this is especially the case if death has occurred quite rapidly. In these cases, probably lesions of the cardiac nerves and ganglia arise, which can only be demonstrated with the greatest difficulty. On the

other hand, however, there is found, especially in those cases in which the agony has lasted for some time, and in which improved conditions have varied with renewed collapse, the grossest changes in the heart muscle, fatty degeneration of the almost complete muscular structure, gross changes in the nuclei of the muscles, and complete fragmentation or hemorrhagic dissemination of the muscular tissue and with this adhering thrombi which have most probably occurred during life.

But it is not the heart alone that is endangered; the **kidneys** are no less affected, as may be noted from the appearance of marked albuminuria and the morphotic constituents in the urine. It is well known that scarcely any case of severe diphtheritic infection runs its course without pathological implication of the kidneys, so that, for example, Aufrecht has reached the conviction that the disease of the kidneys and the plugging of the uriniferous tubules by shreds of tissue, as the result of parenchymatous changes, represent the primary condition of the severe symptom-complex, the heart being simultaneously affected. This may be true of a number of cases, but certainly not of all of them, as the renal affection and the cardiac implication may run their course independently of each other, a condition that I have observed in numerous cases. In this we must certainly agree with Aufrecht that the severest cases are mostly those in which cardiac phenomena simultaneously run their course with pathologic urinary changes, and that the presence of large quantities of albumin in the urine, and, above all, the finding of certain morphotic constituents, makes the prognosis of the cases decidedly more unfavorable. The presence of numerous peculiar, coarsely granular, changed, refractive, decomposed epithelial, cell masses in the urine for a long time has been the sign of an especially unfavorable prognosis. We hardly know what to think when these severe diphtheritic renal changes, which were described long *prior* to the introduction of serum therapy, are now ascribed to the employment of serum.

*The renal affection* also shows itself clinically by a diminution in the amount of urine and is especially characterized in that but few, individual cases excepted, marked *renal hemorrhages* occur in diphtheritic affection, in contrast to the renal inflammations occurring in scarlatina, in which the occurrence of blood in the urine is one of the most important clinical manifestations of the disease. The diphtheritic renal affection is further characterized by the *very rare occurrence of dropsy and the uremic symptom-complex*. The latter condition probably does not arise, in that in severe forms of diphtheria which have not received proper treatment the deadly toxins of the organism caused by the diphtheritic virus develop more rapidly than the picture of uremia with convulsions and coma.

The anatomical changes which are the foundation of the clinical symptoms of the renal condition have been carefully studied and it has been shown that they are due to a true toxic destruction of the kidney in which the macroscopical finding varies greatly. Sometimes the kidneys are hyperemic, enlarged and soft, at other times they are anemic, or the hyperemia is limited to the

medullary substance which may assume a bluish-black appearance; often a moderate distribution of the cortical substance, with a reddish-gray or more grayish-white or finally yellowish discoloration of the same is noted. Microscopically, all stages of degenerative change of the epithelia, cloudy swelling, vacuolar degeneration, necrosis and fatty degeneration is seen in the kidney, so that the nuclear staining property in many areas has been completely lost. What is especially conspicuous, in contrast to the scarlatinal renal changes, is the *focal necrosis* of the epithelia. There are also found, peculiar net-like mass-arrangements of colloid substances in the region of the urinary tubules, thickening and proliferation of the epithelium of Bowman's capsule and loss and disappearance of the glomerular loops, so that, in fact, all portions of the tissue of the kidney are implicated in the malignant process.

We have thus seen two organs affected in a deleterious manner by the influence of the diphtheritic virus, but we still meet certain phenomena in the clinical picture which cause us to devote some attention to a third organ, to *the nervous system*.

In the clinical history just communicated, we have already met with a peculiar paralysis of the velum of the palate and a paralysis of deglutition. I shall relate the history of another little patient aged four years, that showed the phenomena of **diphtheritic paralysis** to a much greater extent than the one whose history was previously related.

The child is treated upon the fourth day of the disease, but already shows a very markedly developed laryngeal stenosis, widely distributed diphtheritic coating of the entire pharynx, tonsils, uvula, and velum palatinum. The child, that had high fever, shortly after admission, had to be intubated, simultaneously after 3,000 antitoxin units of Behring's curative serum had been employed. The course was not favorable in the first days of observation, as a distressing cough constantly forced the tube out and intubation had to be repeated several times; the fever also continued, with temperature between 100.4° F. and 102.2° F., with quite a rapid pulse, up to 150 beats per minute. Marked fetor was present in the child from the onset, and the loosening and destruction of the membrane occurred but slowly and with great difficulty; severe diphtheritic rhinitis, otitis media, with a purulent flow, bronchopneumonic symptoms, complicated the clinical picture; with this there was albuminuria and the signs of cardiac weakness, the latter showing itself with a poor and weak pulse. In this case, as in the other, upon the eleventh day of observation in the hospital, the *first signs of paralysis* appeared in the form of *difficulty in deglutition*. Fluid produced cough, and was regurgitated from the nose; at the same time marked diminution of the patellar reflex appeared. These phenomena developed further and the difficulty in deglutition, simultaneously with newly appearing marked phenomena of *stenosis of the larynx*, compelled us, finally, to desist from renewing intubation, and to perform tracheotomy. After that the fluid could no longer be swallowed, the little patient was nourished three or four times daily with the stomach tube. The following was then noted: The child was very weak; there was still quite high



fever, and the lungs were not quite free, showing the seat of bronchopneumonic areas. Above all, upon observing the child closely, it is noted that the angle of the mouth on both sides droops flaccidly, which gives the face a peculiarly relaxed expression; apparently both *facial nerves* are paralyzed, and with this there is bilateral ptosis, somewhat more marked on the right side than upon the left, and the pupils will not respond to light; apparently bilateral oculo-motor paralysis is present. Whether both sixth nerves are paralyzed cannot be determined, as the child does not clearly perceive light. Further, the velum of the palate is rigid; the difficulty in deglutition has already been referred to. If an attempt is made to raise the child to a sitting posture it collapses so that a well-developed *paralysis of the extensor muscles of the back* must be assumed. The abdominal reflexes are absent, as well as the patella tendon reflex and the phenomenon of the Achillo-tendon. What is, however, more conspicuous than anything else is the peculiar, helpless manner of coughing, and the difficult, hindered respiration, although the cannula is quite free, this is due apparently to an *insufficiency of the entire musculature of respiration* including the *diaphragm*. How dangerous this serious condition of the poor patient is, need scarcely be emphasized. It is evident that death may occur at any moment by suffocation caused by *paralysis of the muscles of respiration*, to which cardiac paralysis may be very readily added. Fortunately, these very well distributed paralyses are rare, they were even rare prior to the employment of serum treatment; since the introduction of the same, cases of *paralysis of this extent are quite exceptional*, and in the last few years, as far as my experience extends, this is the only case of the kind that I have noticed.

Let us continue the discussion of these forms of paralysis.

Two principal groups may be differentiated: First, the *early paralyses*, which are almost always seen in the severest, usually in the septic cases, or in morbid conditions which are closely allied to sepsis. The paralysis always begins in the velum of the palate, and is accompanied by prostration of the entire organism, and severe adynamic-cardiac conditions. The cases are mostly fatal. The second group includes the *late paralyses*, which have also been designated as *post-diphtheritic*. These occur two to three weeks, even five to six weeks after the onset of the diphtheritic affection. Here also the paralysis begins mostly in the velum of the palate so that speech becomes nasal and deglutition difficult; nevertheless, frequently as a first phenomenon, the absence of the patella reflex is noted, occasionally after an increase had existed some days previously. From this point the paralyses advance with a certain irregularity, however, in the manner that paralysis of the eye-muscles, paralysis of accommodation, also frequently facial paralysis arise relatively frequently, whereas paralysis of the musculature of the trunk and even of the respiratory muscles, especially of the diaphragm, belong to the rarest forms of paralysis. Excellent descriptions of these forms of paralysis are found in medical literature; there are the communications of Maingault, Donders, Duchenne, above all, the excellent self-observation of Hansemann, perhaps

also the collection of the observations made by myself in my book that has been frequently quoted. The paralysis may also include the sensory nerves, so that *anesthesia* and *paresthesia* may accompany the motor paralysis: *the sympathetic system* may also be affected so that paralysis of the musculature of the intestines and bladder may be observed.

The anatomical basis of these paralyses is formed partly by neuritic changes in the peripheral nerves (*toxic neuritis*), partly they may be due to central processes. Severe anatomical lesions of the ganglia have lately been described on several occasions. I should not like to enter upon a discussion of these conditions as they have not as yet been definitely settled.

Still a special variety of paralysis should be mentioned which has actually nothing to do with the post-diphtheritic paralysis and is rarely observed, but which should, nevertheless, be recognized, these are *hemiplegic paralyses*.

We have had an opportunity of noting three such cases. The paralysis may occur suddenly and may throughout take on the character of an apoplectic attack; it is of a well developed hemiplegic character. As an anatomical basis, an embolic affection in the corpus striatum or also a circumscribed hemorrhagic encephalitis has been determined. Here, therefore, the point of origin is not the nervous system but the vascular system. Apparently at one time the expression of the severe alteration of the blood mass by toxic substances circulating in the blood, that give rise to coagulation and, further, the adynamia of the cardiac action which goes hand in hand with the destruction of the heart muscle, which, finally, causes clot-formation in the heart or in the vessels.

We have now learned to recognize serious changes of organs which are due to the toxic substances which have entered the circulation that are produced by Löffler's bacillus, however, we dare not close our eyes to other affections which, although they are not specifically peculiar to diphtheria, nevertheless, are of the greatest importance to the entire course of the disease. To this the *bronchopneumonias* must be mentioned which accompany the difficult pharyngeal affections, also *intestinal disease* with malignant *diarrhea*; further, the *phlegmonous lymphadenitis* of the neck, *otitis media* purulenta, occasionally with an implication of the bone of the mastoid process and even of the internal ear; further, *secondary peritracheal abscesses*, abscesses of the mediastinum, etc.; they have all been observed by us, they all may have contributed toward bringing about the fatal issue to the toxically influenced and severely implicated organism. We cannot at this place enter more fully into these **complications**. With proper treatment instituted at the right time, they will be but rarely observed.

We must, however, acquaint ourselves minutely with the very frequent and very readily appearing *implication of the upper air passages*, the nose, the larynx, the trachea and the bronchi in the diphtheritic process, and the *suffocative phenomena*, the phenomena of *diphtheritic croup*, which are due to this implication. I have already called attention to the **diphtheritic disease of the nose** in some of the case histories that I have related; I have also

described an ugly, sero-fluid secretion, which, as I said, excoriates the upper lip, making it sore and causing swelling. If the nose is more carefully examined in such cases pseudo-membranous coatings of the exact anatomical character of true diphtheritic membrane are met with anteriorly in the neighborhood of the nasal openings; however, if this is not the case the process may, nevertheless, be designated as diphtheritic, as the secretion contains the diphtheria bacillus in plentiful amounts. This may be determined by a simple dry preparation, and in cases in which the investigation is negative, a culture will decide in a few hours. It should be constantly borne in mind that every light-yellow, fluid, irritating nasal secretion which excoriates the margins of the nose and the lips is suspicious of diphtheria; these phenomena should be borne in mind; then it will not occur as it has so often with physicians, that after this condition has been present for some days and the affection has been looked upon as a simple coryza, suddenly they have been surprised by severe suffocative laryngeal symptoms of a croupous nature in children, unfortunately at a time when it was too late to use effective measures, and when even from serum treatment and tracheotomy they could not hope for relief. This is especially the case in small children, in nurslings, and then *latent or larval diphtheria* is spoken of; however, those who understand these conditions see nothing latent or larval in them.

At this point I shall mention those forms of *pseudo-membranous rhinitis* in which croup membranes line the entire nasal cavity, so that they may be removed in large pieces with a syringe or forceps. For the most part they run a more chronic course, and this morbid process only rarely or scarcely ever implicates the pharynx or even the larynx, and, nevertheless, in these membranes and in the nasal secretions, Löffler bacilli have been repeatedly demonstrated. Apparently, in this instance the condition is due to a *much less virulent form of disease*, which runs a subacute or chronic course, being purely local; nevertheless, I should like to insist on the need of care even here, and the exercise of all the caution in prophylaxis and therapy usual in the case of true diphtheria so as not to be surprised by more serious developments. It is well known that Cadet de Gassicourt has described similar, more chronic, local diphtheritic affections also in the pharynx, and we have had an opportunity of seeing them frequently, even some years since. Not very rarely diphtheria begins, and at that in its severest form, with an affection of the nose, and prior to the period of serum therapy the not quite unjustified assumption was prevalent that particularly these varieties of the disease were the most dangerous. This naturally does not always happen now, although it is true that the septic forms of diphtheria also for the most part implicate the nose.

By far the most dangerous symptoms are those which arise from the *larynx*, and every physician who is familiar with the **true croupous diphtheritic laryngeal** symptoms will certainly, no matter how experienced, feel anxiety if these symptoms appear in a child under treatment, or increase in severity. The most marked characteristic of this form of the disease is *hoarseness of the*



voice, the barking, dry, hoarse cough, and the long-drawn, almost hissing or sawing, *inspiratory and expiratory respiratory murmur*. It is certainly not always necessary, even if true diphtheritic membranes are detected in the pharynx, to assume diphtheritic disease of the larynx on account of the hoarse voice and barking cough, for these phenomena may also occur in catarrhal laryngitis; it is a well known fact that a very acute catarrhal laryngitis occurs where there is no sign of diphtheritic disease, and with the same symptoms, without seriously threatening the life of the affected child by suffocation, and for this reason the name *pseudo-croup* has been given to the condition. Nevertheless, we will have every reason to look upon hoarseness in a well-defined diphtheritic laryngeal affection with the greatest suspicion and care, and, even in those cases where the pharynx is found completely free, it is considered good practice, either by bacteriological cultural investigation of the laryngeal and pharyngeal mucous membranes or by laryngoscopic examination, to determine that the larynx is free of pseudo-membranes; for there are cases enough in which the formation upon the tonsils and pharyngeal mucous membrane is only slight or in which the pseudo-membranous masses have entirely disappeared from these areas, only then the actual diphtheritic laryngitis appearing. As a rule, diphtheritic laryngitis begins in a period when pseudo-membranes are still demonstrable in the pharynx, so that the laryngitis arises as a result of the descent of the pseudo-membranes from the pharynx to the larynx, and Bretonneau has spoken of these downflowing masses. If the diphtheritic laryngitis develops slowly, the voice gradually becomes hoarse and there is heard, besides, a rough scraping tone accompanying the inspiratory prolongation of the respiration, a less prolonged audible expiration. As a rule, the children have fever, which occasionally reaches high ranges, and now in a relatively brief time, the affection develops more and more to the clinical picture which I shall describe in the following clinical history.

Small boy, aged two, taken ill five days previous. It was noted that the boy was well nourished, but, nevertheless, his appearance upon admission showed that he was very ill. The face had an anxious expression; the eyes were widely opened, the alæ of the nose dilated with each respiration during which the mouth was open; simultaneously we noted that with each of the long piping, hissing, respiratory movements the jugular vein became more prominent—also that the thorax, with the assistance of all the inspiratory muscles, the sternocleido, and the pectoral muscles, was markedly raised; we also noted how, upon inspiration, the intercostal spaces were drawn in, a deep furrow forming at the lower border of the thorax, the scrobiculus cordis sinking in deeply and great dyspnea being present. Apparently, there was not sufficient air in the lungs, so that the intercostal spaces and the other soft parts surrounding the thorax sank in on account of the external pressure of air. We noted the sawing, prolonged inspiration, and the hoarse, creaking, barking tone of the cough; with this there was complete aphonia. A glance into the pharynx by no means showed very extended membranes, we were only able to recognize upon the swollen tonsils some grayish-yellow spots and streaks and similar ones perhaps somewhat thicker upon the posterior mucous membrane of the pharyngeal wall. Nevertheless, we could not doubt that we were dealing with *true diphtheritic croup* and it was necessary to relieve the respiratory difficulty, and that as rapidly as possible. In the first place we noted the anxiety of the child, and then the tense radial artery, the hard pulse, the cyanotic condition of the lips, and also the somewhat livid color of the face, these showing us that the

child was nigh unto suffocation and required speedy help. We *intubated* at once, but we had everything in readiness for *tracheotomy* in the case of this small child, meaning immediately to proceed to tracheotomy in case of failure of the intubation.

What would have happened to this child if we had not relieved it in the manner we have just indicated? Without doubt, the dyspnea would have increased, the respiration would have become deeper and more labored, and, assuredly, we should have seen the child tortured with the terrible phenomena of suffocation, raising itself as much as its strength would allow, fighting for air, with a livid, cyanotic face, blue lips, covered with perspiration, with cold, moist extremities. Perhaps under the influence of these very great exertions the respiration might have quieted itself somewhat; it would have become somewhat freer if the child had succeeded in bringing up some mucus or even some shreds of membrane with the severe coughing. Then the child would have collapsed in its bed and slumbered for a brief time; soon, however, the attacks of suffocation would have repeated themselves and the same phenomena would have returned, but not for a long time. The powers are weakened; the child now remains quiet, in apparent slumber, longer and longer, the lips and the color of the body become more bluish, the respiration apparently becomes freer, less audible, less sawing and hissing, the cough desists completely; however, if we examine accurately, we note only a very superficial, no longer complete respiration. The mind is apparently no longer free, the child is in a comatose condition, from which it no longer awakens, but slowly slumbers into the condition from which there is no awakening. This would be the infallible, unalterable termination which, according to our experience, neither internal nor external remedies would be able to ward off. And because we knew this we did not hesitate one moment to interfere.

And the cause of this terrible onward course? A glance into the jars containing anatomical preparations of pseudo-membranes in the larynx is sufficient to explain this. It will be noted how a thick pseudo-membranous mass plasters the entire larynx, completely closing the rima glottidis, and how thence a thick, tube-like, formed and compact pseudo-membrane fills the entire trachea, entering through the bronchi, through the smallest and finest bronchioles, even coating them for wide areas. With this it can be seen that the lungs have collapsed, are thickened, and partly, especially around the borders, markedly dilated, resembling emphysema. This is, therefore, the anatomical finding of *diphtheritic croupous laryngo-tracheo-bronchitis*; this dreadful disease is almost invariably fatal, provided we do not succeed in bringing help to the suffering child by means of mechanical and specific remedial measures. Here, therefore, there is added to the toxic infectious process of diphtheria, a mechanical condition which brings about death by suffocation. Now we can understand why the ancient physicians spoke of synanche, garotillo, of stryptjuka, etc. In fact, of all the dangers of diphtheria that we have learned to recognize up to now, the danger of suffocation, due to the descending croup, is the greatest, and to be all the more feared because it is a daily frequent danger.

Let us devote a few words to some of the symptomatic peculiarities of the clinical picture of croup.

We have laid stress upon the *aphonia*; this symptom is almost always present, but it may be absent, provided the larynx is free from membrane in the region of the vocal cords, more free than the trachea and the bronchi; therefore, the *presence of the voice by no means excludes diphtheritic croup*; and these cases, as a rule, are the most dangerous because the diphtheritic exudation and necrosis have invaded the parenchyma of the lungs more deeply. The pulse, further, requires consideration. For some time attention has been paid to this, and especially to the beginning retardation of the pulse, its slowing with the advancing asphyxia, and *pulsus paradoxus* described by Kussmaul; the phenomenon that causes the pulse to disappear more and more during the long-drawn inspiration has been looked upon as a valuable sign. It is not easy to demonstrate this in the restless child that constantly throws itself about; but it may be distinguished if some attention is paid to this point. In cases in which this phenomenon can be noted, a very severe disturbance of respiration may be concluded, and we should keep ourselves in readiness to immediately combat the dyspnea, because the children are threatened by the danger of suffocation. Not so constant are the signs of the disturbance of circulation in the urine, such as the presence of *albumin*, or of blood, or a diminution in the amount of urine. In the moment of greatest danger to life they are not to be accorded too great value.

I have now called attention to the most important phenomena of the diphtheritic process; I have as yet not spoken of the *various localizations of the diphtheritic process at other points*, i. e., the diphtheritic *cutaneous affections* which appear particularly in areas that are the seat of eczema, that belong to the ugliest and most painful forms of disease, as they enter deeply into the skin from the subcutaneous tissue, causing necrosis and leading to sepsis, then, for example, giving rise to the disturbances which have been designated *dermato-myositis*, which bring about an icteroid condition of the muscle fibers and belong to the most serious processes that are observed; further, the *diphtheritic disease of the genitalia*, especially in little girls; they resemble those of the *external auditory meatus* and of the *middle ear* and the *conjunctival sacs*. These severe lesions are noted in widely distributed diphtheria epidemics, especially among a class of the population that live under bad unhygienic conditions. I have as yet not spoken of *mixed infections of diphtheria*, of the serious *combinations of diphtheria with measles, scarlatina, whooping cough* and other diseases. It is readily understood that on account of the toxicity of the diphtheritic process all these combinations are of the most serious import, and prior to the introduction of serum therapy every one of these complications had greatly increased the mortality among sick children.

These conditions have all improved, although we must not conceal the fact that the danger to children, even under the influence of this powerful active remedy still requires the most watchful care and the most profound knowledge.



## PROGNOSIS

This leads us to the question of the **prognosis** of the disease. Briefly expressed, every phase of diphtheria, no matter how mild, is to be taken seriously, as well for the patient himself, as also on account of the danger of transmission to those about him. However, we must take into consideration that there are in fact dangerous, and less dangerous epidemics, and that the mortality of the disease is subject to many spontaneous variations. Only, we must not depend upon the mildness of an epidemic in an individual case; this might easily lead to very evil consequences.

In general I may state the following regarding the *prognosis*: *The prognosis is the more unfavorable, the more diffuse the local process, the more the pseudo-membranous deposits cover the entire pharynx, especially distributing themselves to the hard palate, the more smeary the membranes appear, the worse their color and the less localized.* Naturally, the implication of the larynx, with stenotic phenomena renders the prognosis decidedly worse; signs of general sepsis, such as marked fetor, diffuse swelling of the submaxillary and post-cervical lymph glands and, finally, hemorrhages from the pseudo-membranes, hemorrhages from the mouth and nose as well as petechiæ, are calculated to render the prognosis grave. *With all these phenomena the prognosis becomes the worse, the later the proper antitoxin serum therapy is employed after the onset of the affection.*

That the prognosis has become entirely different since the introduction of serum therapy, which has been so frequently mentioned by me, and that by means of this remedy we have obtained a factor which ensures certainty beyond our wildest hopes in prophylaxis and in the therapy of the disease, will already have been recognized from my previous remarks. This subject will be entered upon more in detail later.

## DIAGNOSIS

First I shall devote a little attention to the diagnosis of the disease. We might expect that in a disease which is so characteristically developed as a pseudo-membranous affection of the pharynx or of the pseudo-membranous process in the larynx, running its course with dyspnea and signs of suffocation, the diagnosis could not be mistaken. This is true up to a certain degree, and an experienced practitioner who has cultivated the art of inspection will only rarely fall into error or even be placed in a position of doubt. In fact, the clinical finding of the grayish-yellow, or grayish-green, thick, membranous deposits upon and in the pharyngeal tissues, for the most part decides the diagnosis. How certain this is, may best be seen from the fact that in our hospital the decision of transferring patients into the diphtheria division occurs principally from the clinical findings, and I might say, almost never has a case been looked upon by us as diphtheria and brought into the

pavilion in which only diphtheria patients are accepted that was not proven by a later bacteriological investigation to have true diphtheria. Nevertheless, we must not conceal the fact that there are also *obscure cases*, cases of well-developed follicular angina that give rise to doubt, and *these cases require the bacteriological determination of the Löffler bacillus*. The bacteriological examination decides the diagnosis for these cases, which we place in a separate division. Not as if the presence of diphtheria bacilli without a determinable disease of the pharyngeal membrane or the nasal membrane or of the larynx could and would decide us in favor of the diagnosis of diphtheria, but we may be certain of the fact that apparently healthy persons, also children, may carry diphtheria bacilli around with them. These persons are dangerous to others on account of contagion, *but they are not diphtheria patients themselves*. On the other hand, that individual is affected by diphtheria who has the combination of an angenoid mucous membrane infiltration or even of a pseudo-membranous membrane coating in the nose, in the larynx or in the trachea, in which the Löffler bacillus may be microscopically determined or can be recognized in culture. On the other hand, of those that show similar morbid conditions, in whom the demonstration of the Löffler bacillus is not possible, it may be declared with great probability, almost certainty, that they are not ill of diphtheria. The course of these

latter affections that are mostly due to staphylococci and streptococci is differentiated from the true disease in which the diphtheria bacillus is recognizable. In this, therefore, consists the enormous value of the recognition of the Löffler bacillus, and for this reason it is necessary that the physician should be so thoroughly trained in the determination of the bacillus that it is no longer necessary for him to require the proof from another source, even though it may be necessary to erect laboratories of investigation to aid the busy practitioner. Only the physician must himself be master of the methods of investigation, for the examination is easy and may be readily carried out by any physician who has the slightest training. It is only necessary by means of forceps, previously rendered sterile, to take from the pharynx a shred of the pseudo-membrane; this should then be washed in boiled or sterile water, stroked upon Löffler's blood serum, which is readily purchasable, 2 or 3 tubes being used, well closed with sterile cotton, and kept in a place at the temperature of the body from five to six hours; even after this time the characteristic growth of Löffler's bacillus may be macroscopically

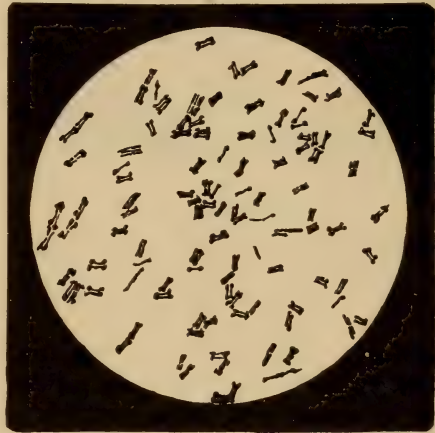


FIG. 33.—PURE CULTURE OF DIPHtheria BACILLI.

determined, which naturally must be confirmed by the microscopic examination of the dry stain preparation (Fig. 33) to determine the presence of the bacillus by its morphological properties. This can be learned and performed by every practitioner; and if this appears too difficult for him he may at least take directly from the pharynx a small particle of the diphtheritic mass, wash it, smear it upon a cover-glass, allow it to dry, and stain it to determine at once the microscopic picture, therefore, to demonstrate the presence of Löffler's bacillus without culture (Fig. 34). Naturally, this second method of examination is not as certain as the first, the culture method; but for many, and even for most cases, it is sufficient. Therefore, there can be no error in the diagnosis, it will be impossible to mistake a scarlatinal angina or a follicular tonsillitis for diphtheria, but also, vice versa, the



FIG. 34.—A PREPARATION FROM A TONSIL-LAR MEMBRANE.

(Containing epithelial cells, leukocytes, mucus threads, staphylococci, streptococci, etc., and characteristic diphtheria bacilli.)

former diseases will not be assumed when actual diphtheria is present, in these cases, to the detriment of the patient, diphtheria being overlooked until too late to treat it with active remedies.

## THERAPY

If there be a desire to become familiar with the entire deplorable helplessness of therapeutic procedures in the cure of a disease, then the history of diphtheria should be studied. Apart from *tracheotomy*, which was introduced into the therapy of diphtheritic croup by Bretonneau, Trousseau, and Guersant, and *intubation* first attempted by Bouchut and finally perfected by O'Dwyer to relieve laryngeal stenosis, the entire therapy of diphtheria, which for many hundred years, and even for thousands of years, has been in the hands of physicians, is a complete failure. I may say this, as since the beginning of my professional activity in a large country practice, as well as later in city practice, I have been in constant combat with this terrible disease, and in my hospital for many years, in spite of arrangements for the treatment of disease which could not be surpassed, have had but the most deplorable and discouraging results. Tracheotomy alone excepted, physicians were almost helpless to ameliorate the course of this grave affection. With the best intention to help them, what has not been tried and recommended among local, internal and external remedies in the long years since the more marked reap-



pearance of diphtheria epidemics in the second half of the nineteenth century, with what have not the poor children been tortured! If it were not entirely superfluous, I could report all of these attempts, but it would require pages, and the sum of it all would be—that everything was futile! Even after the discovery of Löffler's bacillus and of its toxins, when professional endeavors were actively directed toward neutralizing the bacillus in the human organism, I said—it is worth while to take a retrospective view of this kind—in a communication that appeared in the year 1892 regarding my therapeutic attempts: "Whether we look upon the diphtheria toxin as a toxalbumin, as an enzyme, or as another kind of chemical body which is produced in the organism itself, at the present time it displays an action which cannot be controlled because we possess no remedy to combat it." And then followed the enumeration of those methods and remedial agents which we have tried in vain. During this period of completely recognized helplessness, Behring made his discovery regarding the antitoxic action of serum in animals poisoned by diphtheria. After our former experiences, how skeptically the discovery of Behring was regarded may well be imagined. Step by step, however, our doubts disappeared because of the fortunate results which we, with Dr. Aronson, one of the former assistants of the hospital, attained with an excellent curative serum taken from horses. Then followed now, at first tentatively, but with increasing certainty, our communications regarding these fortunate results; I allowed my assistant, at that time Dr. Katz, to report 128 cases at the meeting of the Berlin Medical Society on June 27, 1894; and then, in the year 1895, after the preceding lively discussion in reference to Hansemann's attacks upon serum therapy, I was able to publish 525 cases.<sup>1</sup> I should not have permitted myself to thus call attention to the development of my own observations, if the overwhelming impression of the curative effect of serum therapy were not brought out in such an instructive manner; this forced the previously skeptic Virchow to remark upon consideration of the figures obtained by us that by these "it was sufficiently proven that the curative serum develops a favorable action"—which I would not even admit if it depended upon the proportion of figures, and our own clinical detailed observations did not confirm it, because, particularly in the judgment of therapeutic results, there is nothing more deceptive than mere figures. I shall have to return to this at a later time. But what still further influenced me to refer to my own observations was the curious and rather conflicting fact that in his publications, apparently with definite purpose, Behring himself ignores the results which were gained at our laboratory, and by this curative agency, and scarcely appears to realize that, in spite of all the great honors which distinguished him, the introduction of his curative serum into practice would not have occurred for a long time if it had not received this support.

Regarding the **curative serum** itself, I may refer to Behring's and Ehrlich's publication, the description of its method of production and the deter-

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<sup>1</sup> A. Baginsky, "Die Serumtherapie der Diphtherie," Aug. Hirschwald, Berlin, 1895.

mination of its antitoxic value. We are concerned principally with its method of use. I have published everything that the practical physician should know in my book,<sup>1</sup> have there also mentioned some of the inconveniences which now and then arise from its use, and shall here only refer briefly to the most important points.

The *quantity* of antitoxin units to be employed in the individual case depends upon three conditions: 1, upon the duration of the disease; 2, upon the severity of the affection; 3, upon the age, that is, the size, of the sick child. Therefore, *the largest dose* will have to be used in the case of an older child which is well developed, of about thirteen years of age, and that has been suffering for about three days with general diphtheritic affection, showing phenomena of stenosis of the larynx. Such a case would require the *employment of 3,000 antitoxin units.*<sup>2</sup> On the other hand, in a young child, for example, about two years of age, upon the first or second day of the disease, provided the general implication of the organism be not too marked, not over 1,000 antitoxin units would be necessary. In general it is well to use the full quantity in one injection, the action of the remedy should not be decreased by dividing the dose. Regarding the necessity of using more antitoxin units than was originally thought to be requisite, the observation that fever is still present upon the second day after the employment of the serum will enable us to decide, provided of course that there are no other causes for this fever than the diphtheritic infection, thus, for instance, an enlargement of the glands, pneumonia, etc. The continuance of the symptoms of laryngo-stenosis or the especially slow loosening of the pseudo-membranes, which usually occurs in the first three or four days, *may be the reason for giving another decided dose of 1,000 to 1,500 antitoxin units.* Upon the whole, in the severest cases we have not exceeded *4,000 antitoxin units.* [The most concentrated serum should be employed and care should be taken to procure that made in a well-conducted laboratory and not kept too long in stock.—ED.] The curative serum must naturally be employed with all antiseptic precautions, but scarcely more care is necessary than is required in any other subcutaneous injection. We employ a glass syringe with a butt end of asbestos, and always select the external surface of the thigh as a point of injection. The injection should be given flat, subcutaneously, never too deep, about below the fascia of the muscle and into the muscular tissue. Too deep injections are very readily followed by abscesses.

*The action of the remedy is the more intense and more certain the earlier it is used after the onset of the disease; four to five days after the onset of the disease it loses its action in a great number of cases.*

This we were always able to observe in our cases, although an absolute certainty can never be acquired from figures and much depends upon the circumstances in the individual case. We may regard the fact as assured

<sup>1</sup> A. Baginsky, "Die Serumtherapie der Diphtherie," Aug. Hirschwald, Berlin, 1895.

<sup>2</sup> We have in America resorted to much larger doses. I never thought 20,000 units to be superior in its effects to 5,000.—EDITOR.

that, with the early application of the remedy, neither laryngo-stenotic phenomena nor septic infection of the organism occurs, but that the disease takes on a local non-toxic course soon after defervescence. I must not omit the warning that *the physician should not be too cautious with the dose*, further that he need not fear any *secondary deleterious effects*. All that has been designated by the opponents of serum therapy as evil secondary effects are not to be compared to the extraordinary results of cure, and what may eventually happen is at the most fever lasting two or three days, accompanied with an *urticarial skin affection*, occasionally *arthritic pains*; however, as I can assure any one from my very large experience, these are without importance and without subsequent damage. Truly, bad accidents, provided they cannot be referred to very special conditions—I once saw a uremic attack in a child following an immunizing serum injection after measles—are exceedingly rare; and in the cases mentioned in literature in which *death occurred* this was due to entirely different causes than the use of serum.

The *favorable action of the remedy* may be seen from the following signs: the decline of the temperature up to complete defervescence, and the euphoria going hand in hand with it; the arrest of the exudative and necrotic process at the local focus of the disease; and, even if not in all, nevertheless, in numerous instances, the retardation of the laryngo-stenotic phenomena. This must not all be expected upon the day of the injection; moreover, not rarely an increase of the local process is still observed, but upon the second and certainly upon the third day, if the amount of the injection has been sufficient an arrest of the process is noted and a demarcating redness surrounds the diphtheritic local focus and the disintegration and loosening of the membranes occurs. The peculiar change in color which the membranes undergo is remarkable; from a grayish-yellow to a grayish-green color, to a more light yellow color, which finally becomes a deep yellow. In many cases, and especially in those in whom the serum treatment has been begun upon the second or third day, the danger is over with this antitoxic action of the serum and the patients enter upon an obviously increasing improvement, which becomes more noticeable day by day, leading without interruption to complete recovery.

Let us now devote ourselves to the discussion of the **local treatment** which the diphtheritic areas require, besides the serum treatment. Fortunately, all the former mechanical and chemical measures that were employed to remove the diphtheritic membranes, the use of which tortured the children to a dreadful extent, have disappeared from our therapy. If we still employ these measures it is only to destroy the bacillus and to prevent the production of toxins, as the bacillus is not influenced in its property of life by the serum. For this purpose, gargles of potassium permanganate (2 to 1,000) or boric acid (3 per cent.) may be employed; or a soft cotton brush may be used to paint upon the parts a 1 to 2,000 solution of corrosive sublimate or a combination of ichthyol 5 per cent. with 1 to 2,000 corrosive sublimate, which is especially valuable as ichthyol kills the cocci that are present, whereas corrosive sublimate only influences the bacillus. Finally, powders that are



easily insufflated may be utilized; sozoiodol in combination with milk of sulphur is best for this purpose.

In how far we are able to retain the toxins which are formed by the Löffler bacillus in a nascent condition at the point of their production, even by submucous antitoxin injections, and to neutralize their first effects, as is proposed by Behring lately, I cannot decide, as our experiences have not been sufficient as yet. Reasoning by analogy from experiments in test tubes and from trials with animals, and from results obtained, it is quite possible that unexpected cures may result from this application of the remedy and it is certainly worth a trial.

Besides the measures already mentioned, we employ ice-bags and administer ice internally, which gives decided relief.

It is hardly necessary to state that the patient must receive good nourishment and that it must be administered with care, in so far as this is possible during the febrile period. Small quantities of wine, perhaps also a cinchona decoction, may be administered to maintain strength, and with this the therapy of uncomplicated cases has been exhausted.

The condition in diphtheritic croup and in the sequelæ previously mentioned is different. Diphtheritic laryngeal stenosis requires, as I have already stated, the operation of **intubation** or **tracheotomy**. I mention intubation first, for, in fact, it is preferable to tracheotomy wherever it can be used. The instrumentarium is well known and I have described the manner in which the operation is performed in my book on Diphtheria very completely, besides the text is illustrated by exact photographs. The patients breathe very freely through the intubated tube. It is true, intubation is not a very easy operation and must be practised. In the hands of an unskilled operator, the intubator armed with the tube is a dangerous instrument which may cause death by producing injuries. If the tube has produced damage the children mostly die of secondary bronchopneumonia. This must be remembered and no one should attempt intubation until after thorough practice upon the cadaver. For this purpose I have had a model made which I keep in the post mortem room, upon which I allow my students to practise. The tube by its weight and by friction, especially if it remains in place for some time, brings about injuries which are designated tube ulcers, particularly the lower end of the tube causes this.

With all this, the results of *intubation in connection with serum therapy*, are far more favorable than were formerly attained with tracheotomy. In this connection, I may refer not only to the publications of the skilled American physicians, but also to those of Ranke, Bokai and others; we also, in general, have had very good results, so that among 109 cases reported at the Medical Congress at Moscow, of laryngeal stenosis treated by intubation, only  $9 = 8.73$  per cent. died, and of all laryngeal stenosis 218,  $40 = 15.5$  per cent., a result which, compared with our former figures, is favorable beyond all expectation. These results, as I frankly admit, might even be better if the dexterity of the operating assistants were perfect, but this can hardly be

attained on account of the constant change. From this illustration it can also be seen that mortality figures are dependent upon other causes than the disease itself and the remedies employed to combat it.

*Tracheotomy* can also, as formerly, be used in laryngeal stenosis, but as it causes a wound which is obviated in the case of intubation, and this wound increases the difficulties and dangers, it has attained a secondary importance and is only utilized in cases that are not suitable for intubation, as in the case of a markedly narrow pharynx, etc. Naturally, the results of tracheotomy, which, as a rule, is only utilized as a secondary operation, are only secondary to those of intubation; thus, in 14 cases of primary tracheotomy,  $10 = 71.4$  per cent., and in 22, after repeated intubation of secondary tracheotomies,  $15 = 68.2$  per cent. of fatal cases. Regarding the technique of tracheotomy and the after-treatment, surgical works should be consulted. Proper spray arrangements are also of value in the treatment of diphtheria. One of the most pleasing results of serum treatment is the spontaneous retardation of the stenotic laryngeal symptoms under the action of a properly used spray. Children with symptoms of stenosis of the larynx are allowed to inhale a strong spray of steam for hours, which contains at the most weak solutions of sodium chlorid or a 1 per cent. to 2 per cent. solution of boric acid.

Regarding the **treatment of complications** and *sequels of diphtheria*, we shall first speak of the cardiac affection. Unfortunately, little that is valuable can be said regarding the treatment of cardiac disturbances after they have once become conspicuous. The disturbance of the heart muscle that is due to the toxin of diphtheria can be but slightly influenced therapeutically. All of the heart tonics employed by us, small doses of digitalis, strophanthus, caffein, tincture of the chlorid of iron, etc., have proven unavailing.<sup>1</sup> No remedy has been able to arrest the advancing cardiac death; long rest in bed and good dietetic treatment have shown themselves to be the most valuable remedies in assisting children to withstand this threatening danger. The same is true of nephritis. Careful bland diet, alkaline mineral waters, here and there mild tannin and iron preparations, are almost the only remedies that we employ. Rest and diet act best in this condition, and in summer change of air to a healthy country district is of value. The treatment of other complications, pneumonia, otitis, secondary lymph-gland abscesses, etc., is scarcely different in the case of diphtheria from that arising from other conditions.

In conclusion, a few words regarding **immunization** of children threatened with contagion. In our trials with antitoxin treatment we began at the onset to give prophylactic antitoxin injections to the brothers and sisters of the patients who were brought to us affected by diphtheria, in order to protect them from infection. Fortunately, we were enabled to show in our divisions

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<sup>1</sup> Digitalis should be avoided. Add: fresh and cool air day and night, one or two daily washings with alcohol and water followed by friction, attention to the feet which must not be allowed to be cold, and the hypodermic use of salicylate or benzoate of sodio-caffein in collapse, as taught in another part of this book.—EDITOR.

of the hospital that if by any accident diphtheria were introduced we could always succeed in preventing its dissemination, as was also the case in threatened families. Since then, in a similar manner, immunization has been continued in exposed places and our figures show hundreds protected. Whether, as Behring has lately wished and advised, general immunization of children against diphtheria, similar to vaccination, is to be introduced, or whether it be unnecessary to do this will depend upon the further course of diphtheria epidemics. It is certain that in threatened districts, especially in the country, the further spreading of the disease may be prevented by immunization. On the other hand, in the cases of children that can be well observed and are constantly under the control of physicians, we may content ourselves in employing a sufficient dose of curative serum immediately upon the appearance of the diphtheritic attack. Up to the present our results with serum therapy teach us that early serum treatment is capable of preventing the dangers of the disease. Thus, it will depend perhaps upon the local and social conditions whether the physician is to employ one or the other method of prophylaxis. The dose for immunization purposes varies according to the age and size of the child, between 200 and 300 antitoxin units; only rarely has this dose been increased. As a rule, this has proven sufficient.

I might now close my discussion. I should not like to do this without clearing up a certain point which, unfortunately, serves to bring about a great deal of confusion regarding the value of serum therapy, and thereby incalculable damage to our children, i. e., the application of the statistical method to determine the curative results attained. The **value of statistics** in ascertaining the actual permanence of the processes and circumstances in things and in man is beyond all doubt and incontestable, and the less complicated the relations the more certain and likely will be the results which may be determined from such a compilation, grouping and addition. On the other hand, the scientific and conscientious statistician knows that on account of the complicating circumstances and relations attending figures, the difficulty of their proper estimation grows and the results may be far from the actual truth. What can be more complicated than the course of morbid processes in which certain positive factors as to age, weight, social position, the number of affections, etc., are taken into account, but also innumerable other conditions that cannot be mentioned, even unknown circumstances, such as the constitution of the patient, the nature and virulence of the pathogenic agent, the favorable influence of remedies and of physician, as well as faulty observation and reports, and errors in treatment, may also affect the individual case. This renders professional statistics untrustworthy, and in so far as the most simple relations of figures are not taken into calculation, they are faulty, without value, and harmful. This accounts for the paucity of professional investigations and card index statistics, which scarcely include the simplest and most superficial relations of the investigations, not to mention the details of the individual morbid process. Only after a very large, almost enormous number of results, which include the omissions and errors of individual num-



bers, are observed, is a result attained which approximates the truth. This, above all, renders general statistical reports regarding therapeutic results valueless, and so much inferior to the experience of the faithful observer who notes the minutest details. Hence the useless and detrimental controversy with statistically produced small figures regarding the curative properties of serum therapy. Are the conditions of a single region similar to those of another, or is even one case exactly like another, and even in the same places, under the same physicians, persons, conditions, are the morbid processes exactly similar? Do we not even see in this hospital how the severity of the individual case varies, the children coming to us having entirely different constitutions, their disease and a thousand other conditions varying greatly? But just for this reason the observation of an impartial, well-trained physician who watches with open eyes is more valuable than all statistical reports. From this viewpoint, the judgment of serum therapy arising from careful clinical observations of the special case, with all the variations and surrounding conditions, is the only proper one, and the one that comes nearest the truth. This is the reason why we do not turn to the right nor to the left, but singly and alone, holding to our own base of observation, we arrive at our conclusions regarding the curative value of serum therapy and shall even attain better results.

Let me then say again in regard to the results reached by us, as far as human observation is able to judge, that in no disease of man have we attained such a certain remedial agent as is curative serum in the case of diphtheria. This unalterable opinion is based on our own cases in which we have observed even the slightest variation. To us the individual figure is not merely a number but an actually observed case, and for this reason the sum of the numbers does not confuse us. If in one month the mortality figure of 20 per cent. to 30 per cent. results, and in another only from 5 per cent. to 6 per cent., we can tell exactly why the figures could only be such, and why they could not be different. If in one month a greater number of severe septic cases, or children almost *in extremis* are brought to us with laryngeal stenosis, we know that the majority of these children will be lost and that our mortality reports will be more unfavorable, but this has absolutely nothing to do with the curative remedy, at least not unless we ask that means be resorted to in human beings to bring the dead back to life; for, in fact, in those cases, for the most part, we are dealing with patients in whom death is absolutely certain.

If now while taking into consideration all of these conditions, I say that our mortality figures in those cases which are at all susceptible of cure by the application of curative serum have improved in the proportion of three to one, if I say, further, that by judging the disease forms and patients under similar conditions, our mortality from diphtheria has decreased from about 45 to 15 per hundred, this observation regarding the curative property of the serum appears to me to be more valuable than any number of statistical compilations can be. Do not allow yourselves to be confused by sophistries, do not allow

yourselves to be deterred by terrible pictures which have been described by inexperienced persons who have attempted to use serum treatment, do not allow an hour to go by if you are treating diphtheria patients without resorting to this life-saving remedy.

Finally, to acquaint you with our rules, in a few words which should guide you in your therapeutics at the sick bed of diphtheritic children, I shall give the following:

1. Use of the curative serum in sufficient dose as early as practicable, if possible immediately after the onset of the disease, in doubtful cases even before the diagnosis has been determined by a bacteriological examination.

2. Local treatment of the diphtheritic membranes by mild applications or dusting by some of the remedies previously mentioned, without damaging the mucuous membrane and without unnecessarily torturing the child.

3. Applications of ice-bags to the neck, and internal use of small pellets of ice.

4. Saving and assisting cardiac power by good and proper nourishment and by absolute rest.

5. Early relief of laryngeal stenosis by intubation or tracheotomy.

6. Proper treatment *lege artis* of complications and sequelæ.

# EPIDEMIC PAROTITIS, MUMPS

BY H. FALKENHEIM, KÖNIGSBERG

**CASE HISTORY.**—A boy aged seven, entirely well up to a short time ago. Since three days ago not as active as previously. He began to show pallor, lassitude, he became irritable without definite symptoms of disease making their appearance. The mother then consulted a physician, as the child was very restless the night previously, complaining of earache and because a swelling had appeared upon the left half of the face accompanied by fever.

The swelling is quite conspicuous. It occupies the region in front of the ear, reaches from the zygomatic arch downward to the angle of the jaw and reaches the posterior margin of the branch of the lower jaw to the groove between it and the mastoid process of the temporal bone, forcing the lower half of the ear, particularly the lobe of the ear, outward and somewhat anteriorly. The skin over the swelling appears pale, glistening and tense. The tumor itself has an elastic feel and is somewhat painful upon pressure. The child is hindered in eating, as pains arise in chewing. The tongue is coated, oral mucous membranes slightly reddened. The temperature is somewhat raised, 101.3° F. No other symptoms are discernible in the boy.

The mother declares, upon being asked, that similar cases had not occurred in the child's brothers or sisters, but a playmate of the patient was affected in a similar manner fourteen days previously.

The diagnosis is made by the mother. The boy has mumps, a condition which naturally will get well, but a school certificate is necessary, as the boy in his present condition is not permitted to go to school.

In fact there is a swelling of the parotid gland. The gland is affected in its entirety. In a characteristic manner the swelling produces an alteration in position of the lobe of the ear. The limits of the enlarged gland may be readily determined by palpation. The swelling produces a doughy sensation in the surrounding areas. The nature of the glandular swelling, the absence of inflammatory changes upon the skin which covers it, the previous history, with the report of a similar affection in a playmate, the absence of other symptoms which might produce a swelling of the parotid, make it appear that the diagnosis of the mother is correct. The disease is well known among the laity on account of the peculiar distortion produced by the glandular swelling and the condition becomes particularly noticeable in bilateral disease, the patient assuming a stupid, ludicrous appearance which has led to the disease receiving a number of popular names. It is well known that the disease in the main has a favorable prognosis and in a great majority of the cases does not require treatment.

The patient whose history was just quoted showed a very mild affection, but on account of the fever rest in bed was ordered; to allay the tension of the gland, inunctions of warm oil covered with cotton, and a mild antiseptic mouth wash were ordered to keep the oral cavity clean; a bland fluid diet and a laxative were further administered. No complications having arisen and in particular, as is so frequent, the other side not being affected, the patient was completely well in a few days.

## HISTORY AND ETIOLOGY

The knowledge of parotitis epidemica reaches far back. Hippocrates has given an accurate description of the disease. His experiences completely coin-



cide with ours to-day. The benign character of the disease, the predisposition of youthful individuals, the extraordinary rarity of suppuration of the gland, the secondary inflammation of the testicle, were all known to him. The disease has not changed its character for centuries. It does not appear to be limited to particular climatic conditions. It has been noted in all zones and in all seasons. In the temperate zone it appears that the colder season seems to favor the disease. Thus in 117 epidemics collected by Hirsch, 51 occurred in winter, and among 99 collected by Leichtenstern which were accurately studied, 42 began in the first quarter of the year, 17 in the second, 9 in the third and 31 in the fourth. Prolonged cold and wet weather, sudden appearance of a cold season seem to favor the development of the affection.

The contagiousness of mumps is beyond all doubt. As observations have taught us, transmission of the disease may not only occur at the acme of the infection but even before the glandular enlargement has appeared, and on the other hand, contagion may even occur in convalescence. As a rule, contagion occurs directly from person to person, but the disease may also apparently be conveyed by a third person, even by fomites.

The extension of the disease occurs quite gradually. The contagium distributes itself but slowly and only for short distances. Thus in boarding-schools, at first the inmates of the beds nearest the patient are attacked by the disease and then the disease is arrested, but this may occur, on the other hand, as the result of simple isolation. Often the disease only occurs in a single building, a school, an orphan asylum, a prison, a fortress, and does not occur beyond the walls, disappearing after all predisposed inmates have had the affection without attacking those living beyond these confines. At other times a house epidemic becomes a city epidemic or even affects an entire province. The disease may even obtain pandemic distribution. As in the case of scarlatina, measles, sporadic cases also occur in parotitis epidemica. In some regions mumps appears every year, in other districts only every five or every ten years, again sparing other regions for a much longer period. There are no distinct relations to other infectious diseases, such as to scarlatina, measles, etc. The epidemics may occur in different groups and occur side by side. That children attacked by mumps are not affected by scarlatina, as was observed by Schönlein and Frank, has not been confirmed by later observers. The simultaneous occurrence of mumps with measles, with varicella, with influenza, was noted by Hochsinger a few years ago (*Centralbl. f. Kinderhk.*, 1898, Nr. 12).

The infectiousness varies in different epidemics. While in one the majority of children are attacked, especially if they come in closer contact with each other, thus, for example, in an orphan asylum in Moscow among 300 children, 167 were attacked; in an epidemic observed by Lühe in a cadet school in Ploen, among 131, 118 were attacked—in other epidemics, in families, in children's hospitals, without special isolation, but few cases have occurred, although the inmates had not previously suffered from the disease. The severity of the

affection in different epidemics is also subject to certain variations. In some, though comparatively rare, the cases lose their harmless, ludicrous character, a severe disease appearing with threatening complications.

As a rule, the disease attacks an individual but once, second attacks being exceptional. More frequent attacks than this are extraordinarily rare, particularly in children. Comby saw a girl aged twelve years have an attack of mumps three years after a first attack. Both attacks were unmistakable. Soltmann has seen several such cases and Hochsinger only recently reported cases of this kind.

Children between the ages of five and fifteen are especially liable. Children under two and the aged are rarely attacked. In adolescence the predisposition is still quite marked. There are numerous reports, especially from France, regarding epidemics occurring in barracks. After the fortieth year of life the disease is very rare. The greater predisposition of youthful age, as Leichtenstern remarks, may also be distinctly seen from the fact that in a house epidemic first the younger children, then the older ones and finally, if at all, adults are attacked. In 73 cases Rilliet and Lombard noted the affection in 37 children between five and fifteen years, in 7 between three and four years and none at an earlier age. Soltmann's youngest patient was a year and three quarters old. Steiner's, one and a quarter year old. My youngest patient was only seven months old. It is entirely exceptional that children as young as this are attacked. As a rule nurslings and the newborn are immune to the disease and repeatedly a wet nurse who has been attacked by mumps has been allowed to go on nursing the child, without the child having been attacked by the disease. Gautier observed in an infant, whose mother was attacked by mumps, a swelling of the submaxillary gland. White saw a child six days old and its mother a day later, attacked by parotitis, the mother having a severe affection, while other cases occurred in the neighborhood simultaneously. Homans found, in the child of a woman whose labor came on in the eighth month as the result of parotitis, a swelling of the left parotid gland on the day after birth. The swelling increased upon the two next days and was distinctly painful. These observations permit us to reflect upon the possibility of intrauterine transmission.

Sex does not show a difference in regard to the disease. The apparent predominance of the male sex is due to the greater frequency with which this sex is exposed to contagion, as in schools, cadet schools, barracks, etc. (Comby).

The infection in all probability occurs from the oral cavity and the germs find their way thence to the mouth of the glands. On account of the lack of development of these glands and on account of the narrowness of Steno's duct in nurslings, Soltmann sees the condition which confers immunity at this age of life. Many attempts have been made to discover the nature of the pathogenic agent. Up to the present no uniform result has been obtained. The investigations of Laveran and Catrin deserve attention; these authors detected in the blood, in the parotid and in the testicle, in the fluid of the edema, diplococci. The investigations of Michaelis and Bein, who in two cases found

diplococci in Steno's duct and in the pus of an abscess of the parotid are also noteworthy. These investigations, however, require further proof.

### PATHOLOGY

What *pathological changes* occur in the parotid in mumps have not been determined with certainty. Pure cases are not fatal. As only changes can occur which are susceptible of complete cure it is assumed by analogy that a sero-fibrinous transudation of the periglandular and inter-acinus cellular tissue occurs, whereas the tissue of the gland itself in the main presents a normal condition. Gerhardt saw the diseased gland furnish a secretion which corresponded to the normal. Lombard noted the same condition. Jacob found, in a soldier who was attacked by mumps and who perished on account of edema of the glottis, that the salivary gland was not enlarged, but the cell stratum was filled with a greenish gelatinous fluid, the entire tissue being of a lardaceous consistence. Ranvier proved the absence of inflammatory changes by microscopic investigation. The epithelium of the glandular canal was unchanged, cell proliferation was not present. There was edema of the glottis, edema of the inter-acinous tissue. That, however, in severe cases the gland cells may also show anatomical changes is quite possible. Suppuration occurs with exceeding rarity in parotitis; and then only a part of the gland is destroyed. Although in a portion of the cases the assumption of Leichtenstern may be true, that areas of the gland, as the result of a high grade of swelling, are disturbed in their nutrition and thus destroyed and desquamated by demarcation, in other cases there is only a secondary infection with pyogenic agents from the mouth.

After infection has taken place a *period of incubation* arises, the duration of which has been variously estimated by different authors. In general we may count eighteen days, although somewhat longer periods of incubation (twenty to twenty-two days) are also quite frequent. Demme saw several cases in which the period of incubation lasted but three days. Dukes noted cases in which this was twenty-four days.

### SYMPTOMS

Prodromal symptoms vary in individual epidemics. They may be entirely absent in a number of cases or so slight that they are overlooked and only the swelling of the gland denotes the presence of a disease. In other cases the children become irritable, unwilling to play and are languid. They lose their appetite, their sleep is disturbed, fever occurs, which exceptionally may reach even 104° F. Older children even complain of headache, of fleeting pains in the parotid region, the sensation of a certain tension in the neighborhood of the angle of the jaw upon opening the mouth. The mucous membrane of the mouth shows catarrhal inflammation. Younger children grasp their head and the ear with their hands. The gastro-intestinal symptoms are quite marked



in some epidemics. Soltmann in his youngest patient saw the attack begin with severe fever and an eclamptic attack. Rarely do these prodromes last longer than from one to three days; then with increasing sensitiveness in the parotid, with an increase in tension, with a growing limitation of the movement of the jaw, the swelling in the parotid rapidly appears, most often upon the left side. The gland rises in the groove between the lower jaw and the mastoid process, shows itself as a broad swelling, transversely over the branch of the inferior maxillary and distributes itself in the region in front of the ear. The lower portion of the ear is raised, forced somewhat anteriorly and the readily movable lobe of the ear is almost placed in a horizontal position. In intense cases the swelling advances beyond the immediate surroundings of the parotid. The subcutaneous cellular tissue becomes edematous, infiltrated and has a doughy feeling. The limits of the parotid become indistinct, the swelling in some cases may reach to the orbit, the palpebral space becomes narrowed, the conjunctiva injected. The swelling may distribute itself over the entire cheek up to the angle of the jaw and the submaxillary and sublingual glands may take part in the morbid process, the swelling reaching to the clavicle. The normal division of the neck is then lost, the region of the neck is broadened, and if the disease occurs bilaterally the neck may be broader than the face. Occasionally the parotid as a whole is not attacked at once, but in individual exacerbations. The skin over the swollen area is tense, pale, glistening. The glands are sensitive to pressure. It is noteworthy that in some cases the submaxillary gland, more rarely the sublingual gland, are alone attacked (Penzoldt), whereas the parotid may be spared or only be affected later on (v. Strümpell). These cases gain a certain diagnostic interest, particularly when judging of secondary clinical symptoms after the swelling has disappeared. Under some circumstances in a family one child may be seen with a swelling of the parotid, another child with a swelling of the submaxillary gland.

By means of the enlargement of that portion of the parotid which rests upon the biventer in the retro-maxillary groove, such pressure may be exerted that the resistance of the deep fascia of the neck may be overcome. The lateral wall of the pharynx with the tonsil will then be forced anteriorly and the isthmus of the fauces narrowed. Pressure may be exerted upon the larynx and upon the upper portion of the trachea. Partly as the result of pressure upon the vessels, edematous swelling of the mucous membranes of the pharynx and of the larynx may occur, as also the symptoms of severe laryngeal stenosis. On account of disturbance in respiration in connection with venous hyperemia as the result of pressure upon the veins of the neck, in bilateral, high-graded parotitis, even with moderate fever, cerebral disturbances may occur. The movements of mastication and deglutition as well as speech are made difficult and painful by the swelling. The muscles suffer a functional damage, the enlarged parotid lying as a mass below the branch of the lower jaw, hinders the backward motion of the food in chewing; the mouth can scarcely be opened. Under some circumstances the enlargement may reach so high a grade that

not only solid food but even fluid is refused and the consequences of this become apparent. The proper care of the mouth, on account of insufficient opening of the mouth, cannot be correctly carried out, hence a decidedly fetid breath occurs. Catarrhal stomatitis may appear. Occasionally the surroundings of the mouth of Steno's duct are reddened and swollen and the duct itself may be felt as a stiff band.

The salivary secretion in a number of cases is not altered; in others it is increased or diminished.

Displacement of the posterior lower auditory meatus, swelling of the external auditory meatus, and on the other hand of the Eustachian tube, lead to disturbance of hearing and produce pain which influences the sleep of the little patient.

Often in particularly well marked cases the movements of the head become painful; the head is held rigid and, as a rule, slightly inclined toward the diseased side, besides it is somewhat retracted so as to lessen the tension of the muscles of the head; in a bilateral disease the head is held straight in a medium position, somewhat posteriorly, or even bent somewhat anteriorly.

Occasionally, as the nerve is implicated, facial paralysis is produced. This occurred in the nursling aged seven months mentioned above. DeR were distinct. In six weeks recovery occurred.

The frequency with which the second parotid is affected in the course of the disease varies in different epidemics. The most usual condition, in general, is that a few days after the swelling of one parotid the other is also affected, but this does not occur to the same extent as in the first gland; but epidemics also occur in which it is quite exceptional that the disease is not limited to one side.

Although completely afebrile cases exist, still, as a rule, in the course of parotitis there is fever. Even in the prodromal stage in a great majority of cases this occurs, although the rise in temperature is often slight and only noted in the afternoon, so that in case the other symptoms are not well marked it may readily be overlooked. With the appearance of the swelling of the parotid the fever rises, as a rule, not over 102.2° F., but there are also cases with a decidedly higher febrile course, 104° F., 106.7° F., even lasting for several days and accompanied with prostration, apathy, somnolence and even convulsions, in some epidemics. After a few days, when the parotitis has reached its acme, the temperature falls to normal, occasionally quite suddenly, accompanied with sweating, even before the local process has run its course. The fall in temperature is interrupted and at first a new rise occurs, but not to the former height, if the disease attacks the other side. If relapses occur these renewed rises in temperature are noticeable, as well as with the appearance of complications a decided rise of temperature accompanied with chilly sensations. Thus the fever period, which usually ends in about seven days, may last for fourteen days and even longer.

The pulse frequency corresponds to the temperature in so far as it is not influenced by pain and restlessness.

In severe cases at the height of the disease enlargement of the spleen may be noted (Ewart) also swelling of the cervical lymphatics.

The duration of the disease in uncomplicated cases is in the main dependent upon the intensity of the glandular swelling. In medium severe cases this increases for three or four days and then after edema in the surrounding area becomes less noticeable and the glands are softer, normal conditions are gradually assumed. If the tension of the skin is high graded, bran-like desquamation occurs. In the ordinary cases the process is finished in from eight to fourteen days. In the severer cases this lasts longer and in the abortive cases the process is finished in a few days. In some cases the course is a very protracted and stubborn one. It may take from five to six days until the swelling reaches its acme and if the second gland becomes attacked this period may be even longer, whereas it usually occurs in from one to two days, occasionally after three days.

### COURSE AND TERMINATION

The course and termination of the disease, as a rule, are favorable even in those cases in which complications occur. The parotid returns to the normal although it occasionally happens that for a long time, for the most part in connection with surrounding lymph glands, it remains infiltrated and only gradually returns to normal. Occasionally functional disturbances lasting for some time are observed. Thus Eichhorst saw in a boy aged nine, for three months a decided hypersecretion (cured by atropin). Similar cases were seen by Simon and Prautois; Burton on the other hand saw a diminution in secretion, which was cured by galvanization. Suppurative processes which then only attack a part of the gland are in the main extraordinarily rare. In individual, particularly malignant epidemics, this unusual complication may become frequent, thus in the epidemic at St. Cyr, and in an epidemic observed in Berlin in the spring of 1825 by Hufeland.

The most peculiar complication of parotitis is *disease of the testicle*, which was even known to Hippocrates. This is frequent in men, exceptional in the aged, who in fact rarely suffer from parotitis, and occasionally noted in boys who are near puberty, in the ages of twelve and fourteen years. In earlier life it is extraordinarily rare, a fact to which Laghi called attention. Fabre's patient was nine years, Naumann's eight years. De Cereville saw orchitis in a patient aged four and Steiner even in a nursling, the only case of this kind.

It is interesting that occasionally orchitis may precede parotitis and that at times of mumps, *orchitis parotideae* may occur without parotitis being observed; a complete analogy to the condition occasionally shown in disease of the submaxillary gland in parotitis. The case of Bécclère is characteristic. A boy aged fifteen, in whose school mumps was epidemic, was attacked by orchitis without parotitis, two of his sisters being attacked by parotitis.

The frequency of orchitis varies in individual epidemics of mumps and



the appearance of orchitis is quite independent of the severity of mumps. Rizet even observed an epidemic in which, particularly in the milder cases, orchitis occurred. Patients with gonorrhea, as the observations of military physicians have shown, are by no means particularly predisposed to orchitis.

If orchitis occurs, the testicle usually swells some time between the sixth and ninth days of the disease, whereas the parotitis is already upon the decline, the testicle swelling two to three times its normal size. Usually it is the right testicle. In unilateral parotitis, according to Monti, the left testicle is affected. Simultaneously the temperature rises, often with an initial chill. In the testicle itself, there is a sensation of dull pressure, of tension, also severe pain. Neighboring organs usually remain completely free but they may also be implicated by thickening and swelling. Acute hydrocele may occur, and the scrotum may become edematous and painful. Bilateral disease of the testicle is decidedly rarer than bilateral parotitis. If the second testicle becomes affected this occurs after two to four days; the swelling of the testicle in general lasting from three to six days. This also, as the enlargement of the parotid, as a rule, returns to normal without further consequences; but it must be noted that this condition is not so benign, for particularly according to the reports of French military physicians, in a comparatively large number of cases, atrophy of the testicle has been observed, which for the most part was only partial and not followed by impotence.

Occasionally prostatitis occurs; occasionally also urethritis and cystitis. Rilliet and Sanné noted in a school epidemic among 10 cases of orchitis, 5 with a yellowish tenacious secretion from the urethra.

In what the connection consists between the parotid gland and testicle, which forms the basis for the peculiar implication of the testicle in parotitis, and in what manner orchitis occurs has not yet been cleared up. Orchitis has been noted with particular frequency by military physicians, especially in France, in epidemics occurring in barracks, and this gives rise to the possibility that the germs have been directly transmitted to the urethra, finding their way upward into the testicle—an assumption which finds a certain support in the fact that in a number of cases urethritis is noted, but by no means in all.

In the female the genital tract is also implicated, but by no means so frequently. Besides vulvar vaginitis, Bartholinitis, swelling of the large labia, urethritis, menstrual disturbances, there occurs also, analogous to orchitis, disease of the ovaries. Voigt saw in a girl aged eleven, in the course of mumps, the right ovary swell to the size of a chestnut, being painful upon pressure. The mammary gland may also swell and be painful. Travel observed this disease in a patient aged fifteen, Rizet in a girl aged five. Occasionally in the male, *mastitis parotidea* occurs.

Guelliot noticed a transitory swelling of the thyroid gland. Occasionally, however, rarely, the implication of the tear gland on the one side as well as bilaterally has been noted with swelling of the upper eyelid, so that the tear gland could be felt resembling a hard kernel. Jacob claims to have noted

a pancreatitis occurring acutely and subsiding in a few days, producing a sausage-like tumor, sensitive to pressure below the liver, accompanied by pain in the epigastrium, vomiting, fever (104° F.) in the course of mumps.

*Albuminuria* has been frequently noted and also cases of true acute infectious nephritis, sometimes of a hemorrhagic character (Pratolongo, Isham, Henoch, Croner and others). As a rule recovery occurred.

Among other complications which have occasionally been noted there are to be mentioned, endocarditis and pericarditis (Jaccoud, Grancher and others) terminating in recovery, otitis, osteomyelitis, articular and tendon affections such as occur in the course of scarlatina and gonorrhea, but much more rarely and milder. Occasionally with erythema and various cutaneous affections (erythema nodosum, papulatum, urticaria, petechia). Severe disturbances on the part of the respiratory tract apart from the previously mentioned case of edema of the larynx, from which, per example, Pailhas also lost a boy aged eleven, have rarely been observed; on the part of the digestive apparatus, stubborn vomiting, severe colic, marked diarrhea, finally all terminating favorably.

Milder affections of the same category belong to the picture of mumps, especially if fever is present. On the part of the nervous system there are particularly reports from French observers that occasionally in the course of parotitis and in connection with the same mental disturbances, delirium, convulsions and meningitis occur. These complications occurred in the main in severe, markedly febrile, cases in neuropathic children (Comby), associated with orchitis. Lately Heubner in his text-book has mentioned an interesting case of this kind in which in a strong, intelligent boy aged thirteen a complete psychosis developed with absolute disappearance of memory for former events and forgetfulness of surroundings, which after a period lasting several weeks of sleep and dream life, gradually terminated in recovery. Multiple neuritis (Joffroy) has been noted as after other infectious diseases, and in rare cases besides conjunctivitis severe affections of the eye, keratitis (Zossenstein), iritis (Collomb), retinitis (Hatry), neuritis optica (Tallon) with amaurosis.

It is noteworthy that apart from otitis media, on account of the immediate neighborhood of the diseased parotid gland, disturbances of the ear occur partly without a primary otitis media; severe labyrinthine disease without noteworthy prodromes, with unilateral or bilateral deafness; attacks of vertigo and headache may appear which give but small hope for the return of hearing (Toynbee, Pierce, Alt and others). Although these occurrences are rarer in children than in adults, still several cases have been reported in which children under thirteen have become deaf in the course of mumps.

All of these various complications, mentioned on account of completeness, do not play an important rôle in parotitis. Orchitis is the complication  
κατ' ἐξοχήν.

## PROGNOSIS

The prognosis of the disease upon the whole and in the main may be designated as good, although occasionally epidemics occur in which severe cases

accumulate. Even these in general terminate favorably. Fatal cases are extraordinarily rare. Among 58,331 cases of parotitis which were reported in Denmark from 1870 to 1894 Ringberg (*Jahrb. f. Kinderhk.*, Bd. xlvii, page 313) only found 7 which terminated fatally, among these 3 children (2 in the first year of life). Nevertheless, as Leichtenstern quite properly emphasizes, mumps may become serious in that in scrofulous children it occasionally forms the starting point of serious diseases, particularly glandular suppuration.

## DIAGNOSIS

The diagnosis, as the result of the characteristic symptoms which parotitis furnishes, can scarcely be missed. When the lymph-glands in front of the ear, at the angle of the jaw, begin to enlarge rapidly, the surrounding connective tissue is involved in the inflammatory process and a doughy swelling arises; at the onset, before the skin reddens, before abscess formation becomes plain, the diagnosis of parotitis may be incorrectly made. This occurs, but may be avoided if it be remembered that in parotitis the groove between the mastoid process and the lower jaw becomes filled, a broad tumor forming in this region, that the parotid becomes sensitive to pressure, not, however, so painful as a beginning abscess. A confusion with secondary, so-called, metastatic parotitis is scarcely possible. This is observed in purulent inflammations of the mouth and pharynx, in diphtheria, particularly in severe typhoid, in fact in diseases, especially in acute ones, which go hand in hand with severe prostration, such as acute exanthemata, in puerperal sepsis of the mother and newborn, due for the most part to the entrance of pyogenic organisms from the contaminated mouth through Steno's duct into the gland, partly perhaps also from blood infection. Although here as in parotitis, the parotid gland itself enlarges and in this way the local symptoms show great similarity to mumps, especially as in metastatic parotitis, on account of apathy and somnolence, the pain may not be so marked, nevertheless the presence of the primary affection, the fact that the parotitis occurs upon one side, the slower development, the greater extent of the tumor and the tendency to abscess formation, assist the diagnosis into the proper direction. Difficulties in the clinical picture of mumps only occur when the submaxillary gland or the sublingual gland, without implication of the parotids, swells, or this condition precedes enlargement of the parotid, or if after the glandular swelling has returned to normal, complications, particularly orchitis, must be of value as diagnostic aids.

## THERAPY

The treatment in general is very simple. In a majority of cases covering the gland, to keep out deleterious external substances, application of warm oil, covering with cotton, keeping the mouth clean by rinsing with antiseptic fluids, bland, mostly fluid diet, regulating the bowels, and if fever is present, rest in bed. In a great number of cases the physician is not consulted at all



as the affection is known to be benign. Special attention is to be given to the care of the mouth. If the return to normal of the enlarged gland should be protracted, iodine preparations may be used, per example, inunction with iodine vasogen, painting with iodoform collodion (1-15), etc. If, exceptionally, suppuration occurs the gland is to be incised, great care being taken not to cut the facial nerve branches. In orchitis, rest in bed is necessary, elevation of the diseased organ, the application of cold or if this is not well borne, of moist warmth. All severer symptoms and complications are to be treated according to general principles. The fact that these conditions arise in mumps does not require a change in their management.

After all symptoms have disappeared, a few days after the parotid swelling has returned to normal, the children ought to be bathed. They may then leave the house and come in contact with other children. Although it is correct, that most frequently isolation is practised too late, because professional opinion is not consulted on account of the long period of incubation or the slightness of the affection, nevertheless the patients are to be isolated. In regard to the mildness of mumps, Henoch believes isolation to be unnecessary and Laveran is of the opinion that children should be exposed to the infection to protect them in later life from the complication with orchitis. Although mumps in childhood, in by far the greater number of cases, does not give occasion for the slightest anxiety, still the possibility must be considered that parotitis in youthful individuals, although very rarely, may take a severe course, may be complicated by nephritis, otitis media, etc., and may be the starting point of the appearance of scrofulous phenomena. To this may be added that on account of the limitation of the predisposition to the disease the possibility exists that persons may remain spared from the affection. By immediate isolation of sick children in large institutions, schools, etc., a further distribution of the disease may be prevented. As we are dealing with an infectious disease in mumps, disinfection of all materials that have been in use and thorough cleansing of the sick room is advisable. It is well in regard to the mildness of the disease only to undertake the most necessary measures; all the more, as those about the patient, are not even inclined to do this, if inconvenience or cost be associated with these measures.

#### LITERATURE

- Leichtenstern, "Parotitis epidemica" in "Gerhardt's Handb. d. Kinderkrankh.," 1877, ii, p. 649.  
 Vogel, "Krankheiten der Lippen und Mundhöhle" in "v. Ziemssen's Handb. d. spec. Path. u. Therap.," 1878, vii, 1, p. 91.  
 Longuet, "L'union médicale," 1885.  
 Mettenheimer, "Jahrb. f. Kinderhk.," 1891, p. 383.  
 Marfan, "Rev. mens. des maladies de l'enfance," 1894, p. 420.  
 Saltmann, "Parotitis epidemica" in "Eulenburg's Real-Encykl. d. ges. Heilk." Dritte Aufl., 1898, Bd. xviii, p. 323.  
 Comby, "Traité des maladies de l'enfance," second edition, 1904.

# PERTUSSIS, WHOOPING-COUGH, TUSSIS- CONVULSIVA

BY A. BAGINSKY, BERLIN

IN observing a young child that is coughing, if the cough be accompanied by a peculiar loud, sighing inspiration, several points are to be noted by a careful diagnostician. If the cough is severe, forcible, paroxysm upon paroxysm succeeding one another and finally accompanied by a spasmodic laryngostenotic apnea, so that the face assumes a dusky hue and finally becomes cyanotic, the entire attack showing a threatening character, it is quite evident that such a child is suffering from pertussis, an infectious disease of childhood.

Two points should be noted at once, that a cough of this character presents an affection *sui generis*, and secondly, that such a child should be isolated, as it is suffering from a transmissible disease. This affection of childhood, which apparently presents a severe character, is the purpose of the present monograph.

## GENERAL CLINICAL PICTURE

CASE HISTORY.—A boy, aged four, in whom at first glance no pathologic condition can be noted. The mind is clear, cheeks are red, perhaps somewhat rounder and fuller than is usually noted in children of this age, the child presenting, in general, the appearance of robust health. Pulse and temperature show nothing abnormal, nor does the character of the child's respiration; upon undressing it, however, it appears that the child, apparently in the best of health, does not show a condition of nutrition upon the rest of the body in harmony with the full face, above all, on account of the flaccid condition of its adipose tissue. The child, upon the whole, is not as well nourished as might appear at first glance, the fatty tissue is soft, flabby, the skin is quite dry, almost wrinkled upon the arms, abdomen and legs, and we cannot escape the impression that the face shows a somewhat bloated character, which renders it very probable that the condition of nutrition of the body is not as good as it formerly was.

In searching for the possible reason of this retardation in nutrition, we find that neither the temperature curve nor the condition of the urine nor the feces of the child furnishes a clue; only one thing may give us a point of support for this explanation and this is the report that the child suffers from severe attacks of cough, which are frequently accompanied by vomiting, that these attacks of cough and vomiting occur from four to ten times a day and even more frequently. If the child takes nourishment after an attack of coughing and vomiting of this sort, it may readily occur that the interval between the attacks of cough are not sufficient for complete gastric digestion, that is, for a definite removal of the food from the stomach to the intestinal canal. The parents report that the child has lost weight.

Physical examination shows normal conditions throughout. Everywhere, over the thorax, vesicular respiration, nowhere accompanied by catarrhal râles, nowhere abnormal

dulness; on the contrary, if anything were noteworthy, the decrease in cardiac dulness, which is apparently due to the fact that the pulmonary tissue covers the heart from right to left, showing a lessened *volumen auctum pulmonum* is noticeable. However, the heart sounds are normal and pure; neither in the organs of the abdomen can anything abnormal be determined either by palpation or percussion.

In examining the pharynx and the mucous membranes of the child by means of a tongue depressor, placed over the dorsum of the tongue, the child has an attack of coughing and suffocation.

This attack has some special characteristics. It is noted that the cough is of quite unexpected severity. While the hollow cough becomes more superficial and shorter, paroxysm following paroxysm, the face of the child becomes dark red, the tongue is protruded, the eyes are prominent appearing injected and moist, lacrymation occurring; inspiration during the individual paroxysm of cough is insufficient, the child can scarcely breathe, and only from time to time, between the paroxysms, does a deep, hasty, sighing sound occur, a long continued inspiration; masses of glassy, moist, foaming mucus are brought up; this continues almost up to the point of complete exhaustion. Finally the force of the cough appears broken, the paroxysms become briefer, rarer, looser, the child resumes its normal color and appearance. But not yet completely; apparently the child is disturbed by a new sensation; it appears to suppress a tickling in the throat, it endeavors to swallow to master this; the facial expression becomes somewhat anxious. In fact the cough breaks out anew, this time even more severely than in the first attack. The child can scarcely breathe, so rapidly do the paroxysms of cough follow one another, so short are the pauses which permit inspiration, and now the cough is accompanied by retching, followed by vomiting, which evacuates the contents of the stomach accompanied by masses of glassy mucus, not without continued troublesome retching and occasional attacks of cough. Finally the child is free from cough but in a dreadful condition! Covered with perspiration, exhausted, pale and tired. The pulse has become soft, frequent, over 120 beats per minute. It is plain that this represents a characteristic of the disease, and it is easy to understand that if attacks of this kind occurring from five to six times during the day and perhaps more frequently during the night—for in fact the nocturnal attacks are more frequent and even more severe than those which occur during the day—are repeated, the nutrition of the child may very readily suffer. Thus we understand the contrast between the apparent fullness of the face, which in fact is puffy—it would be too much to say edematous,—and the condition of nutrition of the rest of the body.

## PATHOGENESIS AND ETIOLOGY

Whooping-cough, pertussis or tussis-convulsiva, is a disagreeable disease and one which may extend for weeks and months, finally enfeebling the strongest children, becoming dangerous to younger ones but may even be serious in older children to such an extent that many years may pass before they regain their normal constitutional condition, even if no complicating affections threaten the life in older patients, which may readily be the case.

The disease unquestionably belongs among the infectious diseases; it is not very readily transmitted, but this certainly occurs by contact between children; however, this may take place, as I have observed with certainty, by means of third persons who come in contact with sick children, conveying it to those that were previously healthy. The contagium adheres to rooms, to fomites; it is actually one of those diseases which are readily distributed through a school. In truth the disease spares no age; it occurs in the youngest nursing as well as in adults, although children during the ages from one



to four are most susceptible. I am able to give the following figures from my own observation: Among 2,650 cases admitted to the hospital in ten years the following cases were observed:

830, in the first year of life.  
1,308, one to four years old.  
502, four to ten years old.  
11, ten to fourteen years old.

Although the number up to four years, in general, is somewhat greater than that between four and fourteen, the frequency of the disease during the first four years of life is comparatively much greater, so that there is certainly a predisposition for this age. It will be noted that nurslings are by no means exempt, as was formerly supposed; on the contrary, I have repeatedly had an opportunity of observing, in my consultation practice as well as in the hospital, that nurslings are frequently and very seriously attacked by this affection. Boys are almost as frequently attacked as girls; there is no difference in sex regarding predisposition.

It can hardly be maintained that season and weather influence the frequency of the disease. Whooping-cough occurs in summer to the same extent as in winter, with the difference that the winter epidemics are more tenacious and more serious, as complications on the part of the respiratory organs occur more readily in winter and thus produce a severer form of the disease.

The disease is an infectious, transmissible one.

Much discussion is noted in literature regarding the cause of the disease. The question of a pathogenic agent is discussed as much to-day as at any time; each day bringing fresh investigations, new discoveries—unfortunately too many—and what is even more serious, no two that coincide. I shall only mention, to complete the discussion, that all sorts of schizomycetes, such as fungi that grow upon orange-skins, have been supposed to be related to the disease, that long, anaerobic rods (bacilli) have been looked upon as the pathogenic agent, and that for example, Afanasieff is said to have produced the disease artificially in animals with these rods. I must mention that a specific diplococcus, cultivated from the sputum, has been described as the pathogenic agent and that the numerous authors who have made these discoveries are still discussing the identity of all these findings. Thus, lately, a small pole-bacterium, which occasionally occurs in short chains, described by Arnheim, which appears to be identical with one previously observed by Czajlewski and Hensel in culture and biological behavior; it is quite probable that in numerous cases of whooping-cough this bacterium may be present. Nevertheless there are many doubts regarding these findings, and above all the etiologic importance has not been proved. The relation of all these discoveries becomes even more questionable, since a new bacillus has been lately described by Jochmann, which is said to be closely allied to the influenza bacillus (Pfeiffer), perhaps is identical with it, and, as the author expresses

himself, "must be first considered in investigating the etiology of tussis-convulsiva, as the bacillus is constantly found in the expectoration of whooping-cough during the convulsive stage." I may further mention that Deichler, in the year 1886, found protozoa-like structures in the expectoration of whooping-cough patients, which he believed to be the pathogenic agents; and thus quite a number of microbes have been etiologically suspected from which a choice may be taken according to option, or, what appears to me to be more preferable, it is better to remain a skeptic, or what is even better, every physician should himself search for the concealed, mysterious germ of the disease.

It must not be thought that an attempt has always been made to clear the uncertainty of this curious disease from the standpoint of contagion. Moreover, an attempt was made originally by physiology to search for the causes of the cough, to find the point of development of the disease. We know that the superior laryngeal nerve is the nerve which produces cough and that the posterior wall of the larynx, below the vocal cords, is the region from which most readily, intense cough may be produced. We have learned to recognize the severe reflex cough due to irritation of the nasal mucous membrane in which the fifth nerve is affected, and we were inclined to look upon these points as the actual seats of the pathologic changes which produce whooping-cough; the last mentioned area, all the more, as cases of whooping-cough are observed in which severe attacks of sneezing simultaneously accompany the cough. In fact we are able to observe, and this has been particularly described by Mayer-Huni and v. Herff, that the larynx, particularly, however, the entire posterior portion, the inter-arytenoid mucous membrane, shows a catarrhal condition and inflammatory changes; the nasal mucous membrane and the tracheal mucous membrane, so far as they are accessible to direct investigation, are the seat of quite decided inflammatory irritation and swelling during the course of the disease. Thus there is a certain anatomic basis for the disease—inadequate and unimportant in comparison to the severity of the symptoms—and in connection with our experience of the positive transmissibility of the disease we can hardly explain the condition differently, than by assuming that the disease is due to an infectious germ, causing catarrh of the upper respiratory passages, thus considering whooping-cough among the infectious, catarrhal diseases. Whether any of the previously mentioned microbes, that have been found in the mucous membrane inflammation, are the pathogenic agents or whether we are always dealing with the same one, which I believe to be extremely likely on account of the characteristic nature of the cough, cannot be decided in any direction at present.

Thus it is noticed how etiologic investigation, begun at the bedside, searching for the cause of a disease, leads us from clinical observation; this is quite natural in dealing with an infectious disease, the etiology of which has not been cleared up, for we are actuated by the thought, that with the discovery of the cause of the disease, the possibility of combating and controlling the affection will be attained.

## INDIVIDUAL SYMPTOMS AND COURSE

The character of the disease, in the history just quoted, was uncomplicated; but particularly for this reason it was calculated to show the nature of true, uninfluenced whooping-cough. It was noted that the actual attack of cough was capable of division into two parts, that the first was followed by a second one, and I may mention that sometimes a third follows the second. This is characteristic of whooping-cough and the repetition of the attack is called "reprise," or whoop; this hardly occurs in any other form of cough and from this alone whooping-cough may be recognized from other severe attacks of cough; thus from those due to enlarged bronchial glands, or that form of cough due to adenoid vegetations in the naso-pharyngeal space, etc. Whooping-cough, as a rule, does not at once begin with severe attacks of cough, still less with the characteristic "reprise." The disease develops as a simple catarrh and this is also the mode in which the affection began in the patient whose history has just been quoted. However, from the beginning the cough has an extraordinarily severe character; there is a peculiar, intense, severe irritation which brings about the cough so that the children become dark-red in the face while coughing, even if but few paroxysms of cough follow the irritation. There are no physical signs in the thorax which correspond to this severe cough. The respiratory conditions are normal, there are no râles or other signs of catarrh. More and more these irritative phenomena increase, becoming more severe, so that a few attacks of cough no longer relieve them and paroxysms of cough develop so that an actual attack occurs. Simultaneously the individual attack which now gradually takes on the character of the reprise just described, brings up a mucous, glassy, foaming secretion, quite in contrast to the usual condition in children who, as is well known, scarcely ever expectorate sputum.

Thus we speak of a *first (primary) catarrhal stage* of whooping-cough. Soon other symptoms are added which impress upon these attacks of cough a severe, threatening character. It will be observed that children under the influence of these expiratory attacks of cough gradually suffer for want of air, there is insufficient time for inspiration, moreover it appears as if expiration will not cease, due to the paroxysms of cough which constantly become shorter and more superficial. The face, which is more and more turgid, becomes dark-red, soon even cyanotic; the lips bluish, the small vessels of the conjunctival mucous membrane appear filled with blood, the veins of the face, of the temples and of the neck stand out prominently, like dark bands filled with blood; finally, respiration ceases entirely with the cough, in deepest expiratory position the thorax rests in apnea; finally, with a loud, sighing, whistling sound a new inspiration occurs and slowly, with profuse expectoration of mucus masses and vomiting of the contents of the stomach, deep inspiratory movements occur, interrupted by attacks of cough, completing the first part of the attack, followed by a second and even a third of almost like severity and of the same character.



This respiratory spasm and the apnea which are due apparently to the deepest expiration with closed glottis, have impressed the name *tussis-convulsiva* to the cough in this stage of its development, and the phase of the cough has been designated as the *convulsive stage*. Quite properly, for there are a number of cases in which in the same phase of the cough, during the apnea and in connection with laryngospastic conditions, actual clonic and tonic spasms occur of a severe epileptiform character with complete loss of consciousness and all the phenomena which go to make up the eclamptic symptom-complex. With these symptoms whooping-cough has reached the acme of its development; nor is there a limit for the number of the daily and nocturnal individual attacks. For weeks the cough may remain of the same grade of severity and 30-40-60 attacks may occur in the afflicted children which have a deleterious effect upon their nutrition, exhausting their strength, until unfortunately not so rarely, the organism succumbs to these conditions, or complications severely threaten the youthful life. It is true many recover, probably the majority of children affected even with the severest forms of the disease. However, among those who recover there are not a few who for life suffer from chronic invalidism, showing exhaustive conditions on the part of the heart, such as dilatation of the cardiac ventricles, emphysema of the lungs, bronchiectasis with chronic catarrh, and bronchial adenopathy, besides all varieties of neurotic conditions and many other sequels which will be considered later on.

Finally, when the severity of the disease begins to decline, either spontaneously or from the influence of therapeutic or hygienic measures, true catarrhal symptoms become more and more prominent; the attacks of cough become less severe, the spasmodic symptoms less frequent and milder, the tenacious sputum more yellowish, nummular, purulent—*sputum coctum* of the ancients—whereas simultaneously catarrhal phenomena of all kinds may be noted in the chest by the presence of profuse râles, denoting the development of the *second catarrhal stage*. The attacks of cough become rarer and rarer, appearing only now and then in rapidly passing moments, so that after weeks or months, they only indicate that the child has recovered from a severe infectious coughing disease.

From this brief description it may be noted that the character of the disease is a severe one. Before considering the complications and sequels of whooping-cough some of the most prominent symptoms in the pathological picture will be described somewhat more in detail.

In observing a case of whooping-cough it will be noted that with every attack of cough the child has been forced to open the mouth quite wide and to protrude the tongue; in small children this is particularly marked and as they, as a rule, have very sharp and relatively well developed anterior incisor teeth in their lower jaw, the frenum of the tongue is readily injured upon the teeth when the tongue is protruded; thus under the tongue a small *sublingual ulcer* develops, at first showing a fibrinous coating, which for a while was looked upon as a characteristic symptom of whooping-cough. This is, how-

ever, not the case; it is nothing more than the expression of a mechanical disturbance which may occur in any disease accompanied by severe attacks of cough, and has nothing directly in common with whooping-cough and by no means is characteristic of it.

Another condition which is readily noticed is the fact that particularly small children with rickets, with their already deformed soft thoracic skeleton, suffer very severely under the influence of whooping-cough. Under the influence of the intense expiratory excursus the thorax almost appears collapsed during the dyspnea and apnea. It should be remembered that these children are readily attacked by catarrhal diseases of the lungs and bronchi, that they are very liable to laryngospastic symptoms and thus it will be easily understood how great the danger to life in these children when attacked by whooping-cough. As a matter of fact the majority of these poor, weak children succumb, if the disease be at all severe.

Another category of children who do not possess especial resistance of the blood vessels, in pale and weak anemics, the effects of these severe attacks of cough may be noted from numerous effusions of blood. In such patients it will be seen that hemorrhages occur into the conjunctival mucous membrane, into the subcutaneous cellular tissue, even from the ear, and, although rarely, from the bronchial mucous membrane, and from the lungs in the form of hemoptysis; hemorrhages into the brain shall be considered later on, for they also occur. The subconjunctival hemorrhages which occasionally cover the entire eye and alarm the relatives of the child, as the eyes of the child attain a peculiar appearance, rapidly disappear by absorption of the effused blood, the condition being without importance, much less so than the hemorrhages from the ear or hemoptysis.

It is to be expected *a priori* and is readily understood that in the same manner, as a consequence of the mechanical disturbance from these severe expiratory explosions, other anomalies occur in the infantile body such as *umbilical hernias*, *inguinal hernias*, *prolapse of the rectum*, and general *cutaneous emphysema*, which although quite rare, I have myself observed and is worthy of note. [Intussusception in rare instances.—EDITOR.]

Quite a number of more or less bothersome disturbances complicate whooping-cough without actually having anything in common with the character of the same as an infectious disease; naturally they are not to be looked upon too lightly; and yet they are subordinate to the other complications which develop upon the infectious basis of the disease.

## COMPLICATIONS AND SEQUELS

We shall consider some of the most important of this second group. It will be impossible for me to mention all of the complications which occur but I shall quote from my own experience and from the literature the most prominent of these.

**CASE HISTORY.**—A child, aged one and one-half years, almost moribund, with marked cyanosis even of the hands and finger-nails, dyspnea, in collapse, apparently unconscious, which but a few moments before was attacked by severe convulsions. In this condition the child was brought to the hospital and the physical examination reveals, bilaterally, especially posteriorly upon the left side of the chest, loud diffuse râles, particularly in the scapular region; in the same region bronchial respiration was noted. We were therefore dealing with a severe, diffuse bronchitis and with a large bronchopneumonic focus of the left lung, probably of both lungs. The temperature of the child was not above 101° F. The respiration was superficial and frequent, scarcely countable. The history showed that the child had been suffering from whooping-cough for some weeks; for a few days the symptoms of *inflammation of the lung* had been added, since which time the attacks of cough have become less frequent; in place of this, however, severe convulsions have occurred. We hardly hoped to save the child and this history is quoted to show that bronchitis and *bronchopneumonia* represent the severest danger in pertussis.

The conditions were little better in a second case: A boy aged one year; the history shows that for fourteen days he has suffered from whooping-cough. Three days ago fever began and upon admission to the hospital, a temperature varying between 101° F. and 103° F. was present with excessive dyspnea. In this case diffuse râles also were noted over the entire thorax and a quite extensive area of dullness over which bronchial respiration could be determined was found in the right scapular region. This child also had two general convulsions in one day; the child was unconscious and strabismus was noted. An unfavorable prognosis was given in this case. It is noteworthy that severe attacks of whooping-cough were observed in the child even in this condition.

*Bronchitis* and *bronchopneumonia* are in fact the two complications which develop from the infectious character of whooping-cough, and may perhaps themselves be designated infectious diseases, but when superadded to whooping-cough lend malignancy to the character of the affection and cause the death of young children. The danger in older children is not so great. However, the disease is severe enough and pulmonary inflammations complicating whooping-cough are to be feared under all circumstances; even in older children serious sequels remain: *Atelectases of the lungs* which are difficult to overcome; emphysematous areas at the border of the lungs; chronic bronchitis with a tendency to bronchiectasis, and what is more serious, with a tendency to softening of pulmonary areas under the influence of specific or saprophytic bacteria, such as the tubercle bacillus or the bacillus tetragenus and streptococcus. Thus after years, *bronchiectasis* and *tuberculosis* may end the life of a child particularly when complete convalescence from the original disease has not taken place.

It will be noted that in the first case it was mentioned that the attacks of whooping-cough ceased with the appearance of pneumonia; this is frequently the case but not necessarily so, as is taught by the second case in which the attacks continued in spite of the pneumonia. I have already called attention to the importance of *convulsions*. In such instances they were the accompaniment of the general infection from pneumonia. But even without this, convulsions are serious additions to whooping-cough, and this is true even in older children, for this complication, which is very serious, often in a few days causes the death of the child. Probably here also the general infection is the source of the convulsion, for as we shall learn further on, the con-



tagium of tussis-convulsiva has a toxic-malignant character. Convulsions may also occur from the combined action of several pathogenic agents under the influence of increased temperature.

Convulsions may also occur due to other causes, thus from an accompanying nephritis which gives rise to uremia. Many years ago, in a discussion in the Berlin Medical Society, I called attention to the fact that every infectious disease of childhood, even a subacute or chronic eruption, might be accompanied by *nephritis*; thus nephritis is by no means a rare occurrence in whooping-cough. I have lately had the opportunity of seeing several cases of nephritis occurring in the course of pertussis, fortunately without uremic convulsions.

**CASE HISTORY.**—A well developed girl suffering for a few weeks from a severe attack of whooping-cough frequently accompanied with vomiting. The face is somewhat turgid, the eyelids are swollen so that the bulbi can be seen with difficulty; the internal organs present nothing that is pathologic, however, the child is feverish, the temperature varying between 99.5° F. and 104° F.; the composition of the scant urine is conspicuous in that on some days it does not amount to more than 130 c.c. with a specific gravity of 1,020, on other days amounting to 400 c.c. The urine is turbid, of a light brownish red color, containing albumin, some few blood corpuscles, large numbers of casts mostly hyaline, beside numerous leukocytes. This presents a form of acute nephritis which could not be more typical than as a sequel of scarlatina. It is quite obvious that such a nephritis may also present a uremic symptom-complex, and I have noted this condition in a similar case in which a fatal termination occurred. The case was one of a well-nourished strong girl aged three who developed severe convulsions in connection with a marked case of whooping-cough; the spasms recurred frequently and did not yield to sedatives nor to venesection. The child hardly had an opportunity of recovering from its soporose condition, partly due to the influence of the dreadful attacks of whooping-cough which recurred twenty times daily and were accompanied by convulsions, partly also on account of uremic intoxication; for three weeks we had an opportunity of observing this case, which finally terminated fatally on account of the repeated convulsive attacks. Unfortunately it was impossible for us to obtain an autopsy, so that the question had to remain open as to whether certain changes of the brain (hydrocephalus?) were responsible for the lethal outcome.

From this it is evident that nephritis represents a serious complication of pertussis and that we have good reason for devoting our attention to this complication.

Certain *anomalies of the heart* also require observation; in my text-book I mentioned the occurrence of sudden death due to cardiac collapse and unquestionably changes of the heart muscle play a certain rôle in this condition. If the cases are observed, which succumb after the disease has lasted a long time, degeneration of the heart muscle can often be noted. This was described by Silbermann many years ago. I have also observed this condition, but the minute study of these degenerative changes has not yet been concluded. It is evident that besides the probable direct toxic action of the poison of whooping-cough upon the heart muscle, the powerful stasis in the right heart which is due to the attacks of cough, is capable of damaging the heart muscle; all the more so if the children, under the influence of frequent vomiting and insuffi-

cient nourishment, suffer more and more in their nutrition, becoming feeble and anemic.

In the course and in connection with *tussis-convulsiva* the most remarkable and rare *diseases of the nervous system* occur which require careful consideration. They are not common, but I am able to speak from my own experience and report some very peculiar pathologic conditions.

The first case which called my attention to the peculiar disturbance of the nervous system, most probably due to the toxic effect of the poison of whooping-cough, was the case of the three year old daughter of a lawyer, who was brought to me in a most remarkable condition. A robust, strong child who had suffered from severe whooping-cough for weeks, suddenly presented a condition—I can scarcely express it differently—resembling the brute creation. Everything that characterizes a human being had disappeared in the child. Without speech, without sight and hearing, without taste, the child snapped at everything with which it came in contact in a stupid and insane manner; senseless and uninfluenced by its surroundings the child gave forth roaring sounds, feeling its way from place to place, running into everything and without the slightest sense of perception; even the property of mastication was lost and only with the greatest difficulty could the poor, unfortunate child, which had almost become brutal, be nourished. In this pitiable condition the child remained for weeks and only very gradually did a return to normal occur in that signs of the sense of perception by sound, curiously showing itself in attention to music, then of smell and sight reappeared. Gradually the sensorium became free so that the child showed a sense of recognition for its surroundings. After the ice had once been broken the return to a normal psychical condition was surprisingly rapid, and I was able to return the child to the happy father completely cured. The child showed no motor disturbance. The entire process affected the sensory and psychical spheres.

I observed two cases in the hospital which were not quite so characteristic but nevertheless important; I have mentioned them briefly in my text-book but shall be a little more explicit at this point.

A boy aged three and a half, well nourished, was admitted in January, 1897. Three weeks before his admission to the hospital he had suffered from attacks of whooping-cough, which prior to admission were combined with general spasms (convulsions). Upon the day of admission there was high fever, almost 104° F., which disappeared in the next few days so that the temperature dropped to normal. In the following night severe spasms occurred beginning upon the left side, in the region of the facial nerve, and affecting the nerves of the arm, soon distributing themselves to the right side and finally, after a severe attack of cough, disappearing. After a number of relatively good days in connection with attacks of cough, severe crying spells occurred, apparently produced by hallucinations of fear, the child assuming an expression of decided fear, constantly exclaiming "to bed, to bed," whereas in fact he was in bed. Attempts at quieting him were entirely ineffectual; only very gradually did he become calmer; however, the exclamations of fear and cries repeated themselves during the night; finally, the child slept, awoke upon the next morning with a clear mind and remained so in the further course of the whooping-cough, which gradually disappeared; the child remained free from similar attacks. The child was discharged cured.

In this case we were dealing with a psychical alteration closely allied to the well known *pavor nocturnus* of children, only with the difference that the terror did not occur during the night.

The third case closely resembled the first one described and occurred in a girl aged two, admitted in December, 1897. Prior to admission the child suffered for six weeks from whooping-cough accompanied by convulsions; under the influence of these conditions the child lost the sense of recognition for its surroundings and for this reason was brought to the hospital. The child was well nourished but showed itself as completely unconscious of its surroundings. The eyes, with the wide staring pupils were directed into space; with this there was rigidity of the muscles of the neck with slight opisthotonus; spasm in the somewhat spastic upper extremities and in the fingers, movements of mastication, deep sighing and occasional moaning, and loud screaming. The child had a normal temperature, pulse being from 96 to 100 in a minute. During the next days the child showed a more and more almost brutal, stupid condition. Bleating with suppressed voice; unmotivated movements of resistance with hands and feet, with a disturbed position of the extremities when a more quiet condition occurred; taking of nourishment in a greedy, hasty manner; complete apathy for the surroundings so that the child when taken up stared; when placed in bed it bored its face in the pillows while the trunk and buttocks were directed upward almost in an irregular knee-elbow position. Very gradually and slowly did the condition of the child improve and the mind become freer so that the child occasionally sat up and showed some sense of perception for its surroundings; when asked to shake hands it only slowly and hesitatingly complied "as if a veil covered the consciousness of the child," as the history remarked. With this the facial expression appeared serious and not childlike. Finally, the child became pleased with the ticking of a clock; slowly and with isolated sounds did speech return, at least to the extent that the child repeated words, speech up to then having disappeared completely. With this improvement of the mind the attacks of whooping-cough which had persisted and which were still quite severe, finally ceased and the child was discharged from the hospital in a greatly improved condition. The case recovered completely.

The similarity of this case to the one first reported is obvious, and as it is not likely that any serious anatomic lesion was the foundation of these peculiar psychic alterations, which, however, finally disappeared with relative rapidity, we may either consider an edematous transudation of the cerebral hemispheres due to the influence of the severe attacks of cough, or a toxic influence of the brain due to the toxins of whooping-cough as the source of the nervous psychical disturbances. It is true in the last case, at the onset of the disease, there were certain symptoms pointing to a meningo-encephalitic irritation, such as rigidity of the muscles of the back of the neck, spasm, etc.; but on the other hand fever was absent so that a marked inflammatory infection of the meninges was not likely.

Nevertheless the cases are quite remarkable and medical literature is not rich in similar communications; however, other cases are mentioned which may be counted in this category and will aid to impress some of our observations.

Thus in literature there is a case of *sudden blindness* reported by Alexander and the author regards the cause of this condition to be due to a meningeal irritation produced by cerebral edema and so, also, other cases of disturbance in sight, due to probable cortical lesions of the brain, characterized as sensory affections under the picture of "soul blindness."



It is not surprising that under the influence of these severe attacks of cough, occasional *hemorrhages may occur into the brain* with the characteristic symptoms of *cerebral apoplexy*, hemiplegia, loss of consciousness with succeeding spastic unilateral paralyses, which only very gradually but nevertheless completely disappeared without producing a permanent damage to the affected child, but nevertheless are only with great difficulty corrected by orthopedic manipulation.

Stranger than these forms of paralysis are others due to serious lesions of individual areas of the brain and spinal cord, which either terminate fatally or produce a permanent damage to the infantile organism.

Thus I saw a case of *typical, ascending, Landry's paralysis* with a rapid, fatal termination in a boy, occurring in connection with whooping-cough; I still see him before me, a pale boy about seven years old, brought to my office, who collapsed completely in attempting to walk after a few staggering steps, and in the next few days being completely paralyzed with exquisite symptoms of a bulbar affection giving rise to difficulty in deglutition and respiration; thus there are individual cases of *spastic spinal paralysis*, of *encephalitis with paraplegic symptoms*, of *multiple sclerosis*, with beginning *bulbar symptoms* and later *spastic paralytic condition*, of *polyneuritis* with parasthesia, anesthesia and paralysis.

These sequels upon the whole are isolated when compared with the great frequency of whooping-cough in general, but they are calculated to show the affection as a serious and dangerous one.

I may refer, for other cases from my clinic, to an article by one of my assistants (May) in the *Archiv für Kinderheilkunde* in regard to nervous affections of this form.

In what manner these complications occur is entirely unknown on account of our lack of knowledge of the pathogenic agent of whooping-cough; it is hardly possible to refer all these conditions alone to mechanical causes, hemorrhages, edema and the like, and we are forced to think, the more such cases are observed, that similar to the condition in diphtheria, a toxic substance generated by the pathogenic agent circulates in the blood, as in the case of diphtheria, which affects the nervous tissue causing slight repairable, or also severe, unchangeable alterations; with the assumption of a toxic substance we are able to explain the peculiar observation which, as a rule, is noted in whooping-cough, the *hyperleukocytosis*.

## MIXED INFECTIONS

As I have mentioned diphtheria, it is unfortunate that in whooping-cough other infectious diseases can be by no means excluded, that, moreover, combinations of pertussis with other infectious diseases bring about *mixed infections*, this being by no means of rare occurrence; thus we commonly find pertussis in combination with measles in young children, and also with scarlatina and diphtheria; the terrible condition may be imagined of a simultane-

ous diphtheria and whooping-cough in a child in whom, on account of a croupous, laryngostenotic affection a tracheotomy has to be performed, the cannula being kept in place and dreadful attacks of cough occurring—or in a case of measles, which is accompanied by bronchitis, the painful condition of whooping-cough being superadded. It is not to be wondered at, if these children succumb, with the symptoms of exhaustion or if pulmonary atelectasis or bronchopneumonia occur. Among the complicating diseases we also find, and not particularly infrequently in little children, *otitis media*, this being by no means a harmless and painless affection, for it frequently threatens life on account of its general effect when occurring in the course of whooping-cough.

After what has been mentioned it will readily be seen with what a dangerous disease we are dealing in younger children. In our statistics of infant mortality we reckon whooping-cough among those diseases which show a decided influence in the general mortality.

### PROGNOSIS

From the statistics of the city of Berlin, only to mention this one city, we are able to note that in the years from 1890 to 1899 (ten years) the mortality in children from whooping-cough amounted to 4,868; among these more than half, 2,619, occurred in the first year of life. From the mortality tables calculated per thousand, in the population of Berlin, it will be noted that in the years from 1885 to 1896, 9.54 per thousand occurred from pertussis, greater than measles, which only amounted to 6.88 per thousand, and even more than scarlatina, which amounted to 7.83 per thousand. Thus there is every reason for fearing this disease.

The prognosis becomes more serious if complications are added, or preceding disease or constitutional anomalies, influence the health of the child; thus children with old caseous, tuberculous foci are in great danger, as well as children with old pleuritic adhesions, with still existing or previous rickets, syphilis, etc. Above all, the youngest children are threatened and for an infant there is perhaps no more dangerous serious combination than rickets with whooping-cough. It will be noted, from my explanation, that the direct danger to life is not all that is to be considered; that dangers may exist in other directions, in that many children become permanent invalids or only recover with great difficulty, to finally succumb to tuberculosis; many retain pulmonary emphysema, a weakened heart, others nephritis, others all kinds of mechanical lesions such as umbilical hernia, inguinal hernia or nervous affections, paralysis, etc. In a word the prognosis of the disease is always serious.

### DIAGNOSIS

The diagnosis does not give rise to difficulties. At the most, at the onset the disease may be difficult of recognition until the characteristic attacks of

cough with a whoop and vomiting occur. If a genuine attack has ever been noted the diagnosis becomes certain; wherever there is doubt the signs of a severe attack of cough with an almost negative condition in the thorax, and the fact that the attacks occur particularly during the night, which is a very rare condition in simple catarrhal respiratory diseases, will aid the diagnosis. There are two varieties of disease with which whooping-cough may be confused: the cough which accompanies chronic pharyngeal catarrh and adenoid vegetations, and that form of cough produced by bronchial adenopathy. The first of the two affections may be readily recognized by an examination of the pharynx; the second is more difficult; nevertheless, even here, enlargement of the cervical lymph-glands and the physical signs obtained upon auscultation and percussion upon the posterior aspect of the thorax, offer points of support for the diagnosis; in the main, however, attacks of whooping-cough are so characteristic that they are even familiar to laymen and finally the presence of a hyperleukocytosis in doubtful cases will decide in favor of whooping-cough.

### PROPHYLAXIS AND THERAPY

The early recognition of the disease is certainly of great importance. Unfortunately more for the friends and relatives of the patient than for the patient himself; for even with an early diagnosis we are incapable of aborting the cough or influencing its course; but the surroundings of the patient may be protected by removing and isolating the affected individual. This is of especial importance in schools, for we are thus able to prevent the patient from attending school and thus distributing the disease. As a matter of fact the prevention of contact and holding at a distance is the only prophylaxis in whooping-cough; there is no other protection of which I am aware. Even more than the prophylaxis is the therapy of the disease limited, and, as we may frankly admit, helpless.

Up till now there is no active therapy of whooping-cough, although every day, air bubbles of therapeutic successes are exploded. Unfortunately now, more than ever, pharmaceutical industry is busy in forcing remedies upon us; up till now, all without effect! Whether we prescribe reputed specific measures, which are none, like pertussin—a sweetened thymus extract or extract of chestnuts, antibacterial remedies of a general nature such as carbolic acid, thymol, resorcin and many others, whether these remedies are administered internally or externally, no matter, they are and remain without effect. The attempt has been made a hundred times to hang cloths dipped in carbolic acid over the beds of children; occasionally amelioration is said to have occurred; according to my experience it is entirely without result; the same is true of inhalations of oil of turpentine, naphthalin, petroleum, nitrite of potassium, tincture of eucalyptus, gazeol steam, sulphurous acid, etc. This only tortures the children; we accomplish nothing with these remedies, for the disease progresses uninfluenced if it does not even show a deleterious effect, as



occasionally after the use of naphthalin, in that the respiratory organs show an inflammatory reaction and bronchitis develops.

I do not intend to mention the entire army of these useless remedies but only to speak of a few which have a somewhat favorable action. First is the extended use of fresh air; the more we are enabled to allow the children to remain in the fresh air the more readily do they overcome the disease; for this reason the children are sent into the woods, to the seashore and to the mountains, hence it is advisable to send the children to a different locality; unfortunately to the detriment of those localities to which the children are brought as the germs of the disease are carried from place to place. If in a cold climate, during the winter, it is impossible to use the treatment by fresh air we should see to it that the rooms of the children are well ventilated, the child often being removed from one room to another, to keep away the contagium which is distributed in this manner from the surroundings of the child.

Among drugs only two groups are to be considered; the one of a somewhat antifermentative, antizymotic character, and here quinin and quinin derivatives take the front rank. The salts of quinin in doses of 0.1–0.3–0.5 per dose two to three times daily, euchinin, which has no bitter taste in somewhat increased doses ( $1\frac{1}{2}$  grams corresponding in action to 1 gram of the quinin salt) in some cases of whooping-cough have a decided action and serve simultaneously as a tonic, assisting the constitution of the child. With great care the preparations of salicylic acid, such as aspirin, antispasmin, are to be employed, as salicylic acid is capable of exerting a deleterious effect upon the heart muscle.

The second group of remedies includes all those which have a sedative effect and diminish irritation; here the first place is taken by morphia itself, then codein which perhaps is better borne by children than morphia; extract of belladonna, chloral hydrate, phenocollum hydrochloricum and the greatly praised antipyrin which occasionally is of decided effect; finally the entire list of bromin combinations, among which bromoform has lately taken the front rank. Bromoform, according to the age of the child, in from 3 to 5 to 10 drops three times daily, is a convenient method of administration and with some care also a harmless remedy, which is capable of diminishing the attacks of cough and thus perhaps to shorten the duration of the disease. With these remedies, which must be changed frequently, and combined with others, we are able to offer some relief. Of all the other remedies formerly and lately advised I scarcely employ any, nor of the anti-irritative remedies which were advised upon theoretical grounds such as insufflation into the nose with benzol, calomel, boric acid, etc., and I only use these remedies if severe attacks of sneezing accompany the cough.

I need hardly add anything further in the treatment; in severe nervous conditions, in irritative conditions of the brain, meningitis, cerebral hemorrhage and other effusions of blood, in nephritis, in a complicating bronchitis and pneumonia, the remedies usually employed in these conditions are of use here; severe and repeated vomiting requires especial observation and besides

small doses of morphia or codein the preparations of bismuth are capable of ameliorating and perhaps improving the condition. I need not mention nursing nor diet in this disease.

The children should be well nourished, according to their age and power of digestion. Meat juice, tropon, plasmon and foods of this kind may be used from time to time. Finally, after recovery from the disease hygienic measures must be employed to strengthen the child; after recovery a prolonged residence in a warm climate, such as a southern sea-coast, will certainly be of advantage if the physician is able to send his patient there.

[Convulsions must not be permitted to last any length of time. Any attack may be fatal, or at least give rise to an intracranial hemorrhage, with injury to the brain, hemiplegia, etc. Chloroform should be handy and be used at once. Many years ago I published a case in which it was used thirty times a day, and the presence of the physician was required several days in succession, with favorable results.

The author mentions belladonna; but not in the impressive way it deserves. It has been recommended from time to time during the nineteenth century and given up as useless. The reason for this is the faulty method of its administration. I have used it more than half a century, and know of nothing better, or equally as good. Take the case of a child of two years: Give 6 drops of tincture of belladonna, 3 times a day. Unless the drug cause a "feverish" flush on the cheeks within half an hour, which must last half an hour or more, it has no effect. If 6 drops have no such effect, give seven, eight, nine or more every time. The effect must be attained *every time*, 3 times daily. Give as many drops as are required to accomplish that end. After a few days more drops will be required. After about a week the full dose will probably have to be doubled. Throat and pupil symptoms do not annoy the young as they do the adult. If this be done persistently, whooping-cough will last as many weeks as months without it. Moderation of the cough will be observed within two weeks or less. As long as it is severe it will give rise to some complications which the author pictures in his masterly exposition. The saving of those months means the saving of pneumonias, convulsions and acute or chronic or lifelong ailments caused by the treacherous infection. I am quite positive that the omission of an effective treatment of whooping-cough is sinful.

—EDITOR.]





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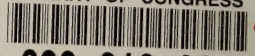








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